The New England
Journal of Medicine

Copyright, 1946, by the Massachusetts Medical Society

Volume 234 MAY 9, 1946 Number 19

A VISIT WITH OSLER*

REGINALD FITZ, M.D.†

BOSTON

THOSE of us who were house officers at the Massachusetts General Hospital during the summer of 1910 will remember that the weather seemed continuously hot and that a great many citizens appeared to have typhoid fever. The medical wards were filled with the disease; during June, July and August we grew almost too familiar with its looks and with the dire manifestations of hemorrhage or perforation. But there were compensations: our sickest patients somehow often managed to grow well; not all hemorrhages were fatal; occasionally our surgical colleagues would sew up a perforated intestine and make us realize the miracle of modern surgical skill; and certainly we learned a great deal about an important infection.

August 25 was a particularly hot day. Even the newspapers commented on the high humidity, and we who were due to work in town over the week end read a little enviously about some of our more fortunate friends who were in the country indulging in the frivolities of tennis or golf. But on that day we had one reward that more than made up for the heat and our worries: Dr. Osler accompanied the visit.

The East Medical Service by tradition is always justifiably proud — proud of its history, of the names on its roster, of the manner in which it does things. At that particular time Dr. F. Gorham Brigham was senior, Dr. William S. Parker was "pup," and I was junior. During the previous four weeks we had been exposed to a new experience. Our regular visiting man, Dr. James J. Minot, had turned over the service to Dr. Joseph H. Pratt. He was then a relatively new importation from Johns Hopkins and seemed fairly to bristle with ideas and energy; among other accomplishments he had gone so far as to introduce the interesting novelty of dictating notes on some of the cases so that our records had in his own words exactly what he thought of them. By and large, to make rounds with him proved an exciting adventure.

On that morning the visit started as usual. We were upstairs in Ward 30 and according to custom had started to move around in clockwise manner when in blew Osler.‡ He came unannounced and unexpectedly. There was no need for any introductions, since Dr. Pratt knew him well and we house officers had heard him speak at the Harvard Medical School on a memorable occasion during the preceding year, so that we were familiar with his appearance — his short figure, his mustache, his keen eyes. Now we were at once delighted by him — in part, perhaps, because to have a distinguished visiting professor from England was by no means a usual happening, even for the East Medical Service; chiefly, because instead of observing the amenities by commenting on the weather or the state of our health, he immediately became part of us by asking, "What have you got here?"

Here was a young woman who had just been transferred from the surgical ward. She had entered the hospital for the removal of a fibroid; everything had gone smoothly for fifteen days, when she was suddenly seized with pain over the right lower chest and with dyspnea and fever. On physical examination, when we first saw her, her heart seemed a little displaced outward, and at the right base below the angle of the scapula was an area of dullness, with distant bronchial breathing and voice transmission that sounded unusually close to the ear. We believed that she had a pulmonary infarct. Dr. Brigham, whose privilege it was to present to the visit each morning the patients with their histories and physical findings, asked Dr. Osler what he thought of the situation. Osler did no more than demonstrate the technic of palpatory percussion by tapping the young woman's back with all the fingers of one hand, and having done so said, "The resistance is quite marked, isn't it?" Dr. Pratt, recalling Oslerian teaching of the importance of increased tactile resistance in the recognition of hydrothorax, asked if he thought the patient had a collection of fluid in her chest. Dr. Osler was noncommittal; all he would say was that the resistance was striking, and finally that it might be well to insert a needle.

*From the East Medical Service of the Massachusetts General Hospital.
†Lecturer on the history of medicine, Harvard Medical School.

‡Dr. Pratt gives a somewhat different account of Dr. Osler's visit (*Boston M. & S. J.* 196:83-89, 1927).

Dr. Brigham performed this operation on the following day and removed 400 cc. of fluid. The patient subsequently developed a recognizable phlebitis in the left leg and later in the right leg, and in the course of time made an uneventful recovery. None of us have forgotten the skill with which Dr. Osler demonstrated so nicely one of the essential signs of fluid, nor the tactful manner in which he suggested its removal.

The next patient had come to the hospital a week before because following a curettage she had begun to lose weight and strength, had become increasingly pale and had finally developed purpuric spots over both lower legs. On physical examination she proved to be feverish and to have a loud murmur at the precordium and a palpable spleen. She was markedly anemic and her blood culture was positive for what the bacteriologists regarded as an atypical pneumococcus. For want of any better treatment she had been given 5 gr. of potassium iodide three times a day, and forty-eight hours later we observed that a painful, peculiar-looking bullous eruption had developed on her face and arms.

When Dr. Osler noticed the rash, he sat down in a chair by the young woman's bedside and apparently paid no attention to her infectious endo-

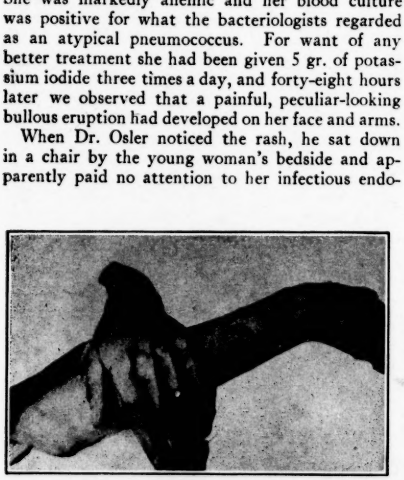


FIGURE 1. *Bullous Eruption after Iodide Administration.*
"Over the backs of the hands and forearms are curious semispherical nodules ranging from 3 to 17 mm. in diameter. The average size is 7 mm. A few are situated in the upper half of the forearms and are symmetrical. Today most of them are infiltrated with blood, have a bright, shining appearance and in color are from light to dark purple. Some are still firm; the larger ones are soft and fluctuant. They were quite tender at the start." (East Medical Records: *note by Dr. Joseph H. Pratt.*)

carditis, in which I, at least, assumed he might be interested. Instead, he began to talk about the rarer skin lesions that may develop following the use of iodides.

I wish that I could remember exactly what he said and how he said it. I am sure that he paid us the compliment of pretending to assume — since we youngsters recently had been welcomed to the company of educated men at a Harvard Commencement — that we were as familiar with medical literature as was he. And I suspect that he began by telling us that here was something curious in the medical line. He may have reminded us of that little known periodical *The New York Medical Gazette and Journal of Health* and of the paper in it by Dr. John O'Rielly — almost invariably misspelt O'Reilly — entitled "On the Effects consequent on the Administration of Iodide of Potassium, illustrated by cases," which appeared in January, 1854. For this makes pleasant reading and tells almost the whole story.

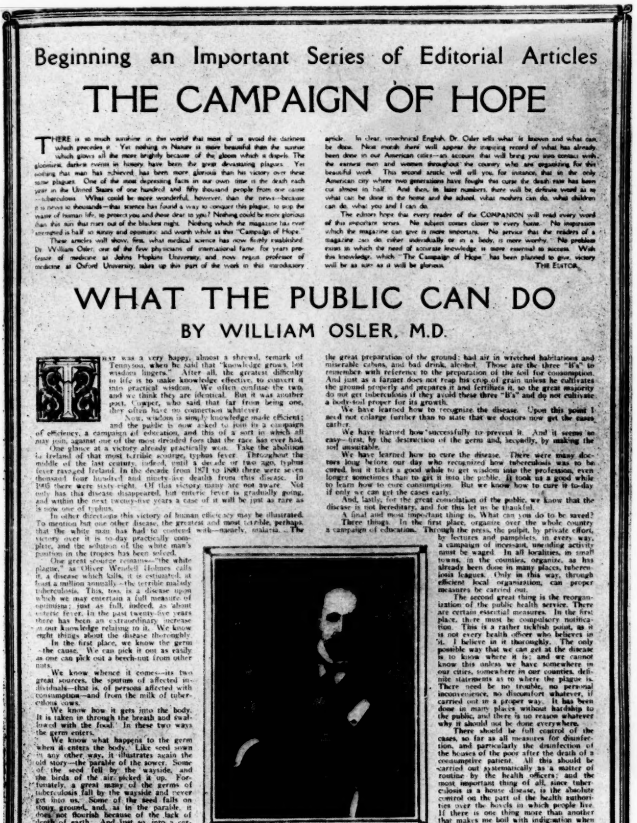
> When a patient has been taking Iodide of Potassium for some days — probably on account of some peculiarity in his constitution — he is suddenly attacked with symptoms of fever, and almost contemporaneously with this disturbance of his system, will be observed on his forehead and face spots, varying in size and irregular in form, of a dark red color. As the case goes on, the spots will be found on the chest and upper extremities, and ultimately will appear on the lower part of the trunk and lower extremities. The spots will gradually become darker, and eventually assume a purple color. After some time, the cuticle will be seen elevated over an odd macula, or filled with a fluid of a purple color; in a short time the cuticle becomes detached, and the subjacent parts, including the true skin and cellular substance will be remarked to be sphaculated to a greater or less extent — as the case progresses, the dead part will be thrown off, leaving ulcers of a considerable depth, or rendering the patient miserable by the total annihilation of an important organ.
>
> With respect to the diagnosis between purpura and the Iodine disease, it will be noted that in the former the maculae appear on the lower extremities first, and that the disease almost invariably occurs in those whose constitutions have been broken down by long sea voyages, or are suffering under some other disease — such as typhus fever, phthisis, anasarca chlorosis, or some other debilitating malady. Again, there is no ptyalism — no bullae — no disagreeable odor — no constitutional derangement — but the grand characteristic distinction consists in the eruption. In the Iodine diseases, like the exanthemata, it shows itself first on the face, while in purpura it appears on the legs.

Or he may have referred to the paper by Hyde that appeared in the *Archives of Dermatology* in October, 1879 and that described all the known cases of a bullous rash developing after the administration of iodides. Not many had been reported, but they all were much alike. This type of rash may develop even after very small doses of the drug have been taken. It usually appears on the face, neck and dorsal surface of the arms and hands, and as a vesicle that rapidly increases in size and may remain discrete or may coalesce with neighboring vesicles to form bullae. In two thirds of Hyde's cases there were cardiac or renal complications, so that he put the question that others have asked since and that has never been answered satisfactorily: Why should patients with heart or kidney disease be predisposed to develop such an unusual type of skin reaction to iodine?

As I remember the scene, Dr. Osler gave the impression of recognizing at a glance an uncommon clinical picture and then went on to show how a good clinician under such circumstances at once begins searching for similar cases. He and Dr. Pratt were soon interchanging a barrage of names and literary allusions that were far over the heads

of the rest of us. And the upshot was that we proceeded to the Treadwell Library to see what we could find.

In those days the Treadwell Library was an ideal working library — a lovely room overlooking down from the walls benignantly at any readers; with a large central table at which one could spread out material to one's heart's content; and with shelves around the room containing enough books and periodicals so that surely one could find all



Beginning an Important Series of Editorial Articles

THE CAMPAIGN OF HOPE

THERE is so much sunshine in the world that most of us avoid the darkness which precedes it. Yet nothing in Nature is more beautiful than the sunrise which grows all the more brightly because of the gloom which it dispels. The gloomiest darkness known in history have been the great devastating plagues. Yet the victory man has achieved has been more glorious than the victory over any some plagues. One of the most depressing facts in our own time is the death each year in the United States of one hundred and fifty thousand people from one cause —tuberculosis. What could be more wonderful, however, than the news—because it is news to thousands—that science has found a way to conquer this plague, to stop the waste of human life, to protect you and those dear to you? Nothing could be more glorious than the sun that rises out of the blackest night. Nothing which the magazine has ever attempted is half so timely and necessary and worth while as the "Campaign of Hope."

These articles will show, first, what medical science has now definitely established. Dr. William Osler, one of the four physicians of international fame for years professor of medicine at Johns Hopkins University, and now regius professor of medicine at Oxford University, takes up this part of the work in this introductory article. In clear, unsentimental English, Dr. Osler tells what is known and what can be done. Next month there will appear the inspiring record of what has already been done in our American cities—an account that will bring you into contact with the earnest men and women throughout the country who are organizing for this beautiful work. This second article will tell you, for example, what the city in American city where two generations have fought this curse the death rate has been cut down to half. And then, in later numbers there will be definite word as to what can be done in the home and the school, what mothers can do, what children can do, what you and I can do.

The editors hope that every reader of the COMPANION will read every word of this important series. No subject comes closer to every home. No impression which the magazine can give is more important. We believe that the readers of a magazine can do either individually or in a body, is more worthy. No problem exists in which the need of accurate knowledge is more essential to success. With this knowledge, which "The Campaign of Hope" has been planned to give, victory will be as sure as it will be glorious.

THE EDITOR.

WHAT THE PUBLIC CAN DO

BY WILLIAM OSLER, M.D.

WHAT was a very happy, almost a shrewd, remark of Tennyson, when he said that 'knowledge grows, but wisdom lingers.' After all, the greatest difficulty in life is to make knowledge effective, to convert it into practical wisdom. We often confuse the two, and we think they are identical. But it was another poet, Cowper, who said that far from being one, they often have no connection whatever.

Now, wisdom is simply knowledge made efficient; and the public is now asked to join in a campaign of efficiency, a campaign of education, and this of a sort in which all may join, against one of the most dreaded foes that the race has ever had.

One glance at a victory already practically won. Take the abolition in Ireland of that most terrible scourge, typhus fever. Throughout the middle of the last century, indeed, until a decade or two ago, typhus fever ravaged Ireland. In the decade from 1871 to 1880 there were seven thousand four hundred and ninety-five deaths from this disease. In 1905 there were sixty-eight. Of this victory many are not aware. Not only has this disease disappeared, but miliary fever is gradually going, and within the next twenty-five years a case of it will be just as rare as is one of typhus.

In other directions this victory of human efficiency may be illustrated. To mention but one other disease, the greatest and most terrible, perhaps, that the white man has had to contend with—namely, malaria. The victory over it is to-day practically complete, and the solution of the white man's position in the tropics has been solved.

One great scourge remains—'the white plague,' as Oliver Wendell Holmes calls it a disease which kills, it is estimated, at least a million annually—the terrible malady tuberculosis. This, too, is a disease upon which we may entertain a full measure of optimism; just as full, indeed, as about enteric fever. In the past twenty-five years there has been an extraordinary increase in our knowledge relating to it. We know right things about the disease thoroughly.

In the first place, we know the germ —the cause. We can pick it out as easily as one can pick out a beech-nut from other nuts.

We know whence it comes—its two great sources, the sputum of affected individuals—that is, of persons affected with consumption—and from the milk of tuberculous cows.

We know how it gets into the body. It is taken in through the breath and swallowed with the food. In these two ways the germ enters.

We know what happens to the germ when it enters the body. Like seed sown in any other way, it illustrates again the old story—the parable of the sower. Some of the seed fell by the wayside, and the birds of the air picked it up. Fortunately, a great many of the germs of tuberculosis fall by the wayside and never get into us. Some of the seed falls on stony ground, and, as in the parable, it does not flourish because of the lack of depth of earth. And just so, into a certain number of us these seeds of tuberculosis enter; but fortunately we are of rocky constitution, and they do not develop. And some of the seed fell among thorns, and the thorns sprang up and choked it. It is a very fortunate thing for some of us that we have a thorny constitution, and when the germs get into us there may be a growth for a short time, and they may thrive and develop, but in a little while thorns spring up. In other words, the constitutional resistance is so great that the germs are killed, and the patient is cured. But, alas! too much falls on good ground, and we know then what happens. It brings forth a hundredfold, and tuberculosis in some form results.

We know how the good ground is prepared. It is well to remember that the seed is not everything—the seed is everywhere, we inhale it every day—it is the soil that is the important thing. Now, how do we prepare the ground for the seed that it may grow to tuberculosis? There are three "baths": Bad food, leading to ill-nutrition, which is the great preparation of the ground; bad air in wretched habitations and miserable cabins, and bad drink, alcohol. Those are the three "ills" to remember with reference to the preparation of the soil for consumption. And just as a farmer does not reap his crop of grain unless he cultivates the ground properly and prepares it and fertilizes it, so the great majority do not get tuberculosis if they avoid these three "ills" and do not cultivate a body-soil proper for its growth.

We have learned how to recognize the disease. Upon this point I need not enlarge further than to state that we doctors now get the cases earlier.

We have learned how successfully to prevent it. And it seems so easy—first, by the destruction of the germ and, incidentally, by making the soil unsuitable.

We have learned how to cure the disease. There were many decades long before our day who recognized how tuberculosis was to be cured, but it takes a good while to get wisdom into the professions, even longer sometimes than to get it into the public. It took us a good while to learn how to cure consumption. But we know how to cure it to-day if only we can get the cases early.

And, lastly, for the great consolation of the public, we know that the disease is not hereditary, and for this let us be thankful.

A final and most important thing to. What can you do to be saved? There things. In the first place, organize over the whole country a campaign of education. Through the press, the pulpit, by private effort, by lectures and pamphlets, in every way, a campaign of incessant, unending activity must be waged. In all localities, in small towns, in the counties, organize, as has already been done in many places, tuberculosis leagues. Only in this way, through efficient local organization, can proper measures be carried out.

The second great thing is the reorganization of the public health services. There are certain essential measures. In the first place, there must be compulsory notification. This is a rather uckish point, as it is not every health officer who believes in it. I believe in it thoroughly. The only possible way that one can get rid of the disease is to know where it is; and we cannot know this unless we have somewhere in our cities, somewhere in our counties, definite statements as to the disease in the house. There need be no trouble, no personal inconvenience, no discomfort whatever, if carried out in a proper way. It has been done in many places, without hardship to the public, and there is no reason whatever why it should not be done everywhere.

Next, there should be full control of the cases, so far as all measures for disinfection, and particularly the disinfection of the houses of the poor after the death of a consumptive. All these should be carried out systematically as a matter of routine by the health officers; and the most important thing of all, since tuberculosis is a house disease, is the absolute control on the part of the health authorities over the hovels in which people live. If there is one thing more than another that makes me lose all with indignation when I visit some countries, it is to see the shocking hovels in which some good fellow-creatures have to live in such wretched dens. This can only be controlled by legal enactments but it is one of the most important of all measures in connection with the crusade against this disease.

Then the third great thing is the proper provision for the care and the cure of the patients.

In the first place, provide hospital accommodation for advanced cases. Not necessarily separate hospitals, but a ward or two wards in the general hospitals to which advanced cases may be sent, and from which they may be cared for—in which they may be looked after until their death. It has been arranged in many countries satisfactorily, why should it not be done everywhere? If possible it is better to have a separate hospital, as has been done in Philadelphia. One of the most valuable benefactions toward this cause is that of [continues on page 621

FIGURE 2. *Title Page of Osler's Article in the February, 1910, Issue of the* Woman's Home Companion (*courtesy of the* New York Public Library).

the hospital yard and in close proximity to the wards; with Stuart portraits at either end looking current literature pertinent to any subject that happened to be of immediate interest, and a good

many ancient books and periodicals that had accumulated with time. It was the pleasantest room in which to work that I have ever known.

Mrs. Myers presided over the library — always an awesome figure to the interns but an old friend of Dr. Osler's. On that morning, for once in our lives, we invaded her precincts with no hint of formality. Mrs. Myers and Dr. Osler greeted each other with distinct marks of affection; and then, as I recall it, Mrs. Myers got out for us to look at certain items dealing with bullous eruptions following the administration of iodide: perhaps Morrow's *Drug Eruptions*, which was first printed in 1887 and has for its colored frontispiece a bizarre example of iodism, or his article in the April and May, 1886 issues of the *Journal of Cutaneous and Venereal Diseases* on the same topic and reproducing the same drawing, or the more recent paper by Gottheil that had appeared in the *Journal of the American Medical Association* a few months previously. We could not find Hare's paper in the 1886 volume of the *New York Medical Record*, which described dynamic dilatation of the aorta; Dr. Osler mentioned this in connection, I presume, with our patient's endocarditis and anemia.

I cannot remember how long Dr. Osler's visit lasted. My recollection is that it was altogether too short and that it ended with a ceremonious parade to the Out Patient Department, headed by Dr. Pratt with Dr. Osler on his right, followed by Dr. Brigham and myself holding ourselves proudly erect, and ending with Dr. Parker, who ran ahead at every possible opportunity to open doors so that his seniors might encounter no physical inconvenience on their march.

Dr. Pratt tells me that a queer postscript should be added to this account of Dr. Osler's visit. Having deposited Dr. Osler and Dr. Pratt in a cab, the East Medical Service resumed its ordinary occupations. Dr. Osler wanted to go to the South Station, and Dr. Pratt accompanied him. Dr. Osler, apparently, disliked to hurry for trains, and so they got to the depot in ample time. They sat together in the stuffiness of a car that had been baked in the trainyard, and so they felt as though they were in a Turkish bath. Dr. Pratt asked Dr. Osler if he knew that one of his papers had been published in the *Woman's Home Companion*. Dr. Osler replied that he had no knowledge of it. Dr. Pratt countered by citing the exact reference. And then Dr. Osler laughed and said, "That explains a great deal!"

A year before, Mr. Bok, editor of the *Ladies' Home Journal*, had asked Dr. Osler to write for the magazine a series of three articles on the American woman that would interest lay readers. Osler had given the matter considerable thought but finally refused, thinking that to do so was a little beneath the dignity of a Regius Professor of Medicine. However, in 1907 at a meeting in Ireland organized by the Countess of Aberdeen he had delivered a lecture, and this later was incorporated as an article in a booklet entitled *Ireland's Crusade Against Tuberculosis*, which had been privately published without copyright. Apparently an ingenious agent in this country had seen this article by Dr. Osler, had realized that he might use it without offending the law, had posed as Dr. Osler's American representative and had submitted it to the *Woman's Home Companion* as an original contribution. Dr. Osler told Dr. Pratt that until then he had never understood the letter of warm expostulation that he had recently received from Mr. Bok.

It is interesting how vividly Dr. Osler and his personality always impressed people who came in contact with him. To those of us who saw him that morning at the Massachusetts General Hospital thirty-five years ago, his visit seems like yesterday, and each recalls something different from it: Dr. Pratt remembers the episode at the South Station, Mrs. Myers still smiles over the memory of the surprised group of house officers as they learned that she and Dr. Osler were old friends, Dr. Brigham remembers his charm, Dr. Parker remembers his demonstration of palpatory percussion in the recognition of hydrothorax, and I subscribe to an interest in medical history that has grown through his influence.

This, I suppose, is the essential secret of successful clinical teaching: so to endow one's associates that each acquires some personal gift in the way of knowledge or approach to clinical problems that is not usual and settled but is inventive and, by adding new rhyme and reason to what was routine, is enduring.

319 Longwood Avenue

MULTIPLE PERITONEAL AUTOTRANSPLANTATION OF SPLENIC TISSUE FOLLOWING TRAUMATIC RUPTURE OF THE SPLEEN*

Report of a Case in an Adult

RICHEY L. WAUGH, M.D.†

BOSTON

THE purpose of this paper is to discuss multiple peritoneal autotransplantation of splenic tissue following traumatic rupture of the spleen and to present a summary of reported cases, together with the first report of a case occurring in an adult.

In the cases hitherto reported, all patients had in youth survived traumatic rupture of the spleen and later, either at reoperation or autopsy, numerous spleenlike nodules were found scattered over the various peritoneal surfaces, including the omentum, small and large intestine, diaphragm and pelvic cavity. Buchbinder and Lipkoff[1] consider the condition a clinical entity and suggest the use of the term "splenosis" to describe it. Despite the similarity, these spleenlike nodules should not be confused with accessory spleens or spleniculi, which are among the congenital anomalies most frequently found at autopsy.

It is apparent from a review of the literature that peritoneal autotransplantation of splenic tissue following traumatic rupture of the spleen is a rare condition. I have been able to find reports of only 15 cases, of which 9 are in the foreign literature and 6 in the American literature. An explanation for the small number of cases thus far reported is given by Hamrick and Bush.[2] These authors refer to the huge over-all mortality rate of between 90 and 100 per cent for traumatic rupture of the spleen, and state that survivals are therefore relatively few in number. Consequently, the chances that such patients will come to abdominal surgical exploration or post-mortem examination are not very great. On the other hand, the actual occurrence of these splenic nodules in patients who have survived traumatic rupture of the spleen may be greater than has been reported.

The first known reference to the condition was that made by von Kuttner[3] in 1910. He found multiple splenic nodules at autopsy in a patient four years after splenectomy following rupture of the spleen due to a gunshot wound of the abdomen. He, however, regarded these nodules as accessory spleens and not as implants of splenic tissue.

Faltin[4] in 1911 was apparently the first to suggest that these splenic nodules were actually splenic implants. His patient was a nine-year-old boy who at the age of three had a ruptured spleen removed following an accident. During the next six years, he suffered with recurrent attacks of abdominal pain, and an operation for chronic appendicitis was performed. At operation, multiple splenic nodules were found scattered throughout the abdominal cavity. One of these was removed, and on microscopic examination it showed the typical structure of spleen, including lymph follicles.

Von Stubenrauch[5] in 1912 reported similar multiple nodules, ranging from the size of hemp seed to that of a pea and with the color of the spleen. These were found on the greater omentum, transverse mesocolon and small intestine in a patient operated on for ileus who ten months previously had received a splenectomy for traumatic rupture of the spleen. Von Stubenrauch concluded that the nodules were a result of so-called "seeding" by splenic pulp, which was scattered throughout the peritoneum at the time of the rupture of the spleen.

In the case reported by Lee[6] in 1923, it was not stated whether the nodules were accessory spleens or implants. Von Küpperman[7] in his report in 1926 called the nodules accessory spleens. Shaw and Shafi[8] in their report in 1937 stated their belief that the nodules were true implants and not accessory spleens.

The 6 cases reported in the American literature include 2 cases reported by Jarcho and Andersen[9] in 1939 and single cases reported by Buchbinder and Lipkoff[1] in 1939, Hamrick and Bush[2] in 1942, Krueger and Mast[10] in 1942 and Boggs[11] in 1945. These reports contain excellent reviews or descriptions of this clinical entity and are well worth reading.

Buchbinder and Lipkoff[1] state that it has been shown experimentally that splenic tissue may be implanted in the peritoneum and readily survives transplantation. Von Stubenrauch[12] removed the spleen in several dogs and seeded the crushed splenic pulp throughout the peritoneal cavities. On sacrificing the dogs one to three months later, he was able to demonstrate the same picture that he had described in man. Marine and Manley[13] in 1920 and Perla[14] in 1936 showed the readiness with which autoplastic splenic transplants grow in rabbits and albino rats, although these transplants were in the abdominal wall and not in the peritoneum.

Hamrick and Bush[2] state that Kreuter[15] in 1920 experimentally excised the spleen in monkeys and smeared the splenic pulp over the peritoneum. Several weeks or months later the animals were sacrificed and splenic nodules were found widely dispersed over the peritoneal cavity. Jarcho and

*From the Surgical Service, United States Marine Hospital.

†Medical director, United States Public Health Service; chief of the Surgical Service, United States Marine Hospital.

Andersen[9] reviewed the literature and found cases of dogs that had had severe abdominal trauma, post-mortem examination revealing nodular implants of splenic tissue scattered throughout the abdominal cavities. These authors also stated that Griffini and Tizzoni[16] as early as 1883 had noted that partial splenectomy in dogs was followed by the development of spleenlike nodules in the peritoneum.

In this connection, Boyd[17] states that in some invertebrates the splenic tissue does not form a definite organ but is scattered beneath the serous coat of the gastrointestinal tract. He also states that some of the higher fishes exhibit splenic tissue divided into separate nodules. Clark[18] states that in fishes, turtles and frogs the spleen exists as an elongated body developed in the mesentery parallel and in close proximity to the digestive tube along the upper, middle or lower part of the intestine.

It therefore appears that the autotransplantation of splenic tissue is comparable to the not infrequent finding of multiple accessory spleens or spleniculi. Moreover, it is apparent that both these conditions represent a reversion to the primitive or atavistic state, in which splenic tissue does not form a definite organ but is strewn throughout the subserous coat of the gastrointestinal tract.

According to the literature, autotransplantation of splenic tissue has been observed only in young patients who have suffered traumatic rupture of the normal spleen. Hamrick and Bush[2] believe that the likelihood of success in grafting cells from splenic pulp on peritoneal surfaces is proportionate to the youthfulness of the normal spleen. These authors state that splenic-pulp cells of young persons probably have properties of increased viability or virulence much greater than those of similar cells in older persons.

The spreading or seeding of splenic-pulp cells on peritoneal surfaces is accomplished through the medium of the free blood present in the abdominal cavity following splenic rupture. After implantation, the nests of cells grow, and the nodules finally become encapsulated by fibrous tissue. On maturity, these splenic implants are found to have histologic structures not unlike that of normal spleen.

According to Hamrick and Bush,[2] parenchymatous cells of no other abdominal organ exhibit similar characteristics except possibly those of the uterine endometrium. The surgeon should keep this in mind and be able to differentiate splenic implants and endometriomas.

No cases have been reported of autoplastic transplantation of splenic tissue following splenectomy for pathologic conditions of the spleen. This is all the more interesting since it has been shown by Jarcho and Andersen[9] that the number of these patients who survive and later come to reoperation or autopsy is much greater than that of patients who survive splenectomy for traumatic rupture of the spleen.

Splenectomy for pathologic conditions of the spleen is usually accomplished without the spreading of splenic pulp over the peritoneal surfaces, but even if under unusual conditions such spreading occurred, the abnormal or diseased condition of splenic-pulp cells would probably hinder successful implantation. This view is also entertained by Shaw and Shafi,[8] who state that the removal of diseased spleens is rarely if ever followed by implants, in spite of occasional tearing with some associated hemorrhage during the excision of a particularly large viscus.

Of the 15 cases reported as showing autoplastic transplantation of splenic tissue, there are only 9 in which the age of the patient at the time of occurrence of the rupture of the spleen is stated (Table 1). The recorded ages ranged from three to fourteen years, with an average of ten years. In striking contrast is a patient who was thirty-two years of age at the time of the rupture of the spleen and splenectomy. A report of the case follows.

A.M.R., a 37-year-old housewife, was admitted on March 14, 1944, for treatment of menorrhagia and metrorrhagia of 5 months' duration. She had an abdominal operation at the age of 28, at which time the appendix and right ovary were removed. At 32 she suffered an abdominal injury in an automobile accident. Immediately after the injury she was taken to a hospital, and 1 hour after admission a splenectomy for traumatic rupture of the spleen was performed. The postoperative course was apparently uneventful.

Three months before the present admission, a cervical polyp was removed on account of menorrhagia and metrorrhagia. This operation, however, gave only temporary relief from bleeding, and the patient noticed an increasing amount of low-abdominal discomfort, fullness and pain. She had had three children, each full term at the time of delivery. The last pregnancy had occurred 12 years previously.

Physical examination revealed a well developed but obese woman who did not appear acutely ill. The skin and mucous membranes of the lips were slightly pale. The temperature, pulse and respirations were normal. The blood pressure was 122/76. Except for the abdominal and pelvic (vaginal) findings, the examination was negative.

The abdomen was obese and flabby. There were two operative scars — one an oblique scar over the left upper quadrant, and the other a midline scar over the lower abdomen. There was moderate tenderness to palpation in both lower quadrants, and a questionable mass in the suprapubic region slightly to the left of the midline. Vaginal examination revealed a cervix with a transverse laceration and a moderate degree of ectropion. There was also a moderate amount of erosion of the cervix, with an area of blanching, apparently at the site of the attachment of the previously removed cervical polyp. The body of the uterus was anterior in position and slightly enlarged. There were no masses or tenderness in the right fornix, but in the left fornix there was a slightly tender and movable mass about the size of a hen's egg. This mass was thought to be either a pedunculated uterine fibroid or an ovarian cyst.

The urine gave a + test for albumin but was otherwise normal. The blood examination revealed a moderate hypochromic anemia, with a red-cell count of 3,400,000, a hemoglobin of 70 per cent and a hematocrit of 34 per cent. The white-cell count was 6400, with 47 per cent lymphocytes, 52 per cent neutrophils and 1 per cent large mononuclears. The sedimentation rate was normal. Blood Kahn and Hinton tests were negative.

Under pontocaine spinal anesthesia, the abdomen was opened through a low midline incision. There were many peritoneal adhesions involving the peritoneum of the anterior abdominal wall, the body of the uterus, the left adnexa and

the sigmoid colon. The uterus was slightly irregular in contour and two or three times normal size. Both tubes and the right ovary were absent. The left ovary was cystic and three times normal size. Scattered over the serous surfaces of several loops of small bowel and the cecum were many small, reddish nodules varying in size from a pinhead to a small pea. The total number of nodules visualized in the limited abdominal wound exposure was about ten. These nodules were believed to be lymphoid tissue due to implantation of splenic pulp at the time of the previous rupture of the spleen and splenectomy. Further exploration of the abdomen by palpation revealed several stones in a flabby gall bladder and a mass of adhesions in the region of the splenic flexure of the colon.

The adhesions of the anterior abdominal wall, omentum, bowel and uterus were separated. A total hysterectomy was done, but the cystic left ovary, the larger cystic portions of which broke during the operative procedure, was not removed. Two small nodules were removed from the serous surface of the small intestine for pathological examination. Chromic catgut was used for ligatures and sutures, and plain catgut for the peritonealization of the pelvic cavity. Three grams of sulfanilamide was sprinkled in the pelvic cavity, and the abdomen was closed in layers with chromic catgut and Kaldermic sutures.

The postoperative course was uneventful, and the temperature was normal on the 5th postoperative day. The wound healed by primary intention, and no vaginal discharge was present on pelvic examination a few days prior to discharge on the 20th postoperative day. The patient was advised to return later for cholecystectomy, but up to August 10, 1945, had not done so.

The pathological report, by Dr. T. F. Crahan, was as follows:

Grossly, there are two small encapsulated tumor masses closely apposed to each other, the larger measuring 2.5 mm. and the smaller 2 mm. in diameter (Figs 1. and 2). On cut section the tumor masses have a slightly reddish color and are soft in consistence, and the larger one shows several small, white follicles measuring less than 1 mm. in diameter. Microscopically, the two masses consist of small portions of splenic tissue completely surrounded by a fairly thick, dense, fibrous capsule. Where they are apposed the fibrous capsule fuses. The larger mass shows malpighian corpuscles, which have prominent arterioles. One of the follicles has a germinal center, with prominent hyperplasia of the reticulum cells. The pulp is extremely cellular, but in some areas definite sinusoids can be made out. Most of the sinusoids are empty, but some contain red blood cells. There is prominent hyperplasia of the sinus endothelial cells and pulp reticulum cells. There are scattered eosinophils and occasional groups of plasma cells. The capsule is focally infiltrated with lymphocytes and

TABLE 1. *Summary of Reported Cases.*

Source	Autoplastic Peritoneal Transplantation of Splenic Tissue						
	DATE	HISTORY OF TRAUMA	AGE AT TIME OF DIAGNOSIS	AGE AT TIME OF RUPTURE AND SPLENECTOMY	REASON FOR EXAMINATION	LOCATION OF NODULES	NUMBER OF NODULES
			yr.	yr.			
Albrecht[19]	1896	Unknown	25	Unknown	Autopsy	Greater omentum, recto-vesical pouch and both domes of diaphragm	Numerous
Schilling[20]	1907	Unknown	47	Unknown	Autopsy	Greater omentum, peritoneum of anterior abdominal wall and pelvis	42 on great omentum (several more elsewhere)
von Küttner[3]	1910	Yes	Unknown	Unknown	Autopsy	Scattered throughout the peritoneum	80–100
Faltin[4]	1911	Yes	9	3	Operation for chronic appendicitis	On loops of large and small intestines	Numerous
von Stubenrauch[5]	1912	Yes	Unknown	Unknown	Operation for ileus	Greater omentum, transverse mesocolon and small intestine	Numerous
Oltmanns[21]	1919	Yes	Unknown	Unknown	Unknown	Throughout peritoneal cavity	Multiple
Lee[6]	1923	Yes	29	14	Operation for ileus	Small intestine and mesentery	Numerous (200–300)
von Küppermann[7]	1936	Yes	15	14	Operation for postoperative hernia	Small and large intestines	100
Shaw and Shafi[8]	1937	Yes	20	Unknown	Autopsy	Abdominal wall, pleural cavity, liver, diaphragm, rectovesical pouch and great omentum	82
Jarcho and Andersen[9]	1939	Yes	8	6	Autopsy	Left dome of diaphragm, liver, stomach, lesser omentum, transverse colon, right kidney, rectum and bladder	Several dozen
	1939	Yes	20	12	Autopsy	Peritoneum, greater omentum, diaphragm and greater curvature of stomach	80
Buchbinder and Lipkoff[1]	1939	Yes	28	8	Operation for pelvic disease	Parietal peritoneum, great omentum, ileum, sigmoid and ascending colon	Numerous
Hamrick and Bush[2]	1940	Yes	13	9	Autopsy	Parietal peritoneum, pelvic cavity, omentum, serosa of large and small intestines and diaphragm	Approximately 75
Krueger and Mast[10]	1942	Yes	16	11	Laparotomy for adhesions	Both surfaces of omentum	20 or 30
Boggs[11]	1945	Yes	14	11	Operation for acute appendicitis	Small intestine	Several
Author	1945	Yes	37	32	Hysterectomy for fibroids	Small intestine and cecum	10 (visualized)

plasma cells, chiefly perivascular. These vessels are mostly thin-walled capillaries. A definite hilus cannot be seen, but in the smaller of the two masses there is a small capillary that seems to communicate with the pulp. The histopathological diagnosis is autoplastic transplantation of splenic tissue.

FIGURE 1. *Photomicrograph of a Full Section of the Two Apposed Splenic Transplants (x 20). Note the capsule, malpighian corpuscles and pulp.*

This case is of unusual interest since the patient was thirty-two years of age at the time of the splenectomy whereas in the previously reported cases the patients were children, the two oldest both being fourteen years of age. This is therefore the first report of peritoneal autotransplantation of splenic tissue occurring in an adult following rupture of the spleen and splenectomy.

SUMMARY

The literature relative to peritoneal autotransplantation of splenic tissue following traumatic rupture of the spleen is reviewed briefly and discussed. Only 15 cases are reported in the literature. Of these, 9 cases are in the foreign literature and 6 cases in the American literature.

An additional case of autotransplantation of splenic tissue is reported and discussed. Its chief interest lies in the fact that the patient was thirty-two years old when splenic rupture occurred and splenectomy was performed.

These peritoneal splenic implants or nodules should not be confused with accessory spleens or spleniculi, which constitute one of the congenital anomalies most frequently found at autopsy, nor should they be confused with endometriomas.

The widespread location of these splenic implants or nodules bears a resemblance to the normal finding in some invertebrates, as well as in some of the higher fishes, in which splenic tissue does not form a definite organ but is scattered beneath the serous coat of the gastrointestinal tract. This analogy therefore suggests that the condition can

FIGURE 2. *Photomicrograph of the Same Section at a Higher Magnification (x 75). This shows two malpighian corpuscles, with lymphoid nodules and small arterial branches. Well formed trabeculae are not seen.*

be considered a reversion to the primitive or atavistic state.

I am indebted to Passed Assistant Surgeon K. M. Endicott, Department of Pathology, National Institute of Health, United States Public Health Service, for his courtesy in making the microphotographs.

References

1. Buchbinder, J. H., and Lipkoff, C. G. Splenosis: multiple peritoneal splenic implants following abdominal injury: report of case and review of literature. *Surgery* 6:927-934, 1939.
2. Hamrick, R. A., and Bush, J. D. Autoplastic transplantation of splenic tissue, in man, following traumatic rupture of spleen. *Ann. Surg.* 115:84-92, 1942.
3. von Küttner, H. Discussion of Ziegler, K. Milzexstirpation und Röntgenbehandlung bei Leukämie. *Berl. klin. Wchnschr.* 47: 1520, 1910.
4. Faltin, R. Milzartige Bildungen im Peritoneum, beobachtet ca. 6 Jahre nach einer wegen Milzruptur vorgenommen Splenektomie. *Deutsche Zschr. f. Chir.* 110:160-175, 1911.
5. von Stubenrauch. Milzregeneration und Milzersatz. *Verhandl. d. deutsch. Gesellsch. f. Chir.* 41:213-215, 1912.
6. Lee, R. T. Survival of splenic tissue after splenectomy. *Lancet* 1: 1312, 1923.
7. von Küppermann, W. Nebenmilzen nach traumatischer Milzruptur. *Zentralbl. f. Chir.* 63:3061, 1936.
8. Shaw, A. F. B., and Shaß, A. Traumatic autoplastic transplantation of splenic tissue in man with observations on late results of splenectomy in 6 cases. *J. Path. & Bact.* 45:215-235, 1937.
9. Jarcho, S., and Andersen, D. H. Traumatic autotransplantation of splenic tissue. *Am. J. Path.* 15:527-546, 1939.
10. Krueger, J. T., and Mast, H. E. Splenic transplants following traumatic rupture of spleen and splenectomy. *Am. J. Surg.* 58: 289-293, 1942.
11. Boggs, E. O. Peritoneal implants of splenic tissue following rupture of spleen. *North. New York M. J.* 2:11, 1945.
12. von Stubenrauch. Verlust und Regeneration der Milz beim Menschen. *Beitr. z. klin. Chir.* 118:285-305, 1919.
13. Marine, D., and Manley, O. T. Homeotransplantation and autotransplantation of spleen in rabbits. *J. Exper. Med.* 32:113-133, 1920.
14. Perla, D. Regeneration of autoplastic splenic transplants. *Am. J. Path.* 12:665-676, 1936.
15. Kreuter. Experimentelle Untersuchungen über die Entstehung der sogenannten Nebenmilzen, insbesondere nach Milzverletzungen. *Beitr. z. klin. Chir.* 118:76-94, 1920.
16. Griffini, L., and Tizzoni, G. Étude expérimentale sur la reproduction partielle de la rate. *Arch. ital. de biol.* 4:303-306, 1883.
17. Boyd, W. *Pathology of Internal Diseases.* Third edition. 874 pp. Philadelphia: Lea and Febiger, 1940. P. 630.
18. Clark, E. R. The lymphatic system. In *Morris's Human Anatomy.* Tenth edition. 1635 pp. Philadelphia: The Blakiston Company, 1942. P. 852.
19. Albrecht, H. Ein Fall von sehr zahlreichen, über das ganze Peritoneum versprengten, Nebenmilzen. *Beitr. z. path. Anat. u. z. allg. Path.* 20:513-527, 1896.
20. Schilling, K. Ueber einen Fall von multiplen Nebenmilzen. *Arch. path. Anat.* 188:65-87, 1907.
21. Oltmanns, C. H. S. Ueber einen Fall von traumatischer Milzruptur mit multiplen Regenerationswucherungen. 36 pp. Halle (Salle), Hohmann, 1919.

GRANULOMA INGUINALE: A PROCTOLOGIC CONSIDERATION*

Joseph Berkowitz, M.D.†

BOSTON

GRANULOMA inguinale was first described as a clinical entity by Conyers and Daniels in 1896, although the etiologic factor was not discovered until 1905 by Donovan at the Madras General Hospital in India. This disease has been considered a venereal one owing to the fact that the lesion is found in most cases on the genitals or scrotum or in the inguinal region, and also because of its frequent association in the same patient with syphilis, chancroid or lymphogranuloma.

The principal pathologic manifestation is found in the skin. The lesion begins as a small papule or macule, which enlarges, ulcerates and spreads by contiguity. The ulceration is covered with pink raised granulations that bleed easily on trauma. Progress of the disease is slow. Over a period of months or years the skin becomes granulomatous and indurated. During this time the patient is ambulatory and in apparently good health, with no evidence of systemic reaction. The pathologic lesion is a chronic inflammatory granuloma characterized by a specific diagnostic cell, a large mononuclear, in the cytoplasm of which are numerous round or rod-like structures known as "Donovan bodies." The preponderance of opinion concerning the nature of these bodies favors the belief that they are gram-negative, nonmotile bacilli in a state of encapsulation.

The incidence of granuloma inguinale is highest in the Negro race and in warm climates. Direct smears from an ulceration or a biopsy specimen from a granulomatous mass are invaluable in the diagnosis. Pathologic entities that present a similar clinical picture and must therefore be ruled out are syphilis, chancroid, tuberculosis, lymphogranuloma and cancer. Specific diagnostic skin tests and laboratory procedures for excluding these diseases are readily available. A specific skin test for the diagnosis of granuloma inguinale has recently been elaborated, but this method has not yet been made generally available. Numerous forms of therapy have been employed, such as x-ray, fulguration and surgical excision, but the best results have been obtained with antimony preparations given intramuscularly.

Case 1. A 26-year-old Negro soldier was admitted to the Surgical Service of this hospital on September 25, 1944, complaining of perianal pain. For 3 months prior to admission he had had pain on defecation, which lasted for 1 hour after completion of the movement. There was no anorectal bleeding, no anal discharge or protrusion, and no change in the size or character of the stool. For 1 week prior to admission there had been severe chafing about the anus, causing pain on walking and soreness on sitting. In 1935 the patient had had gonorrhea, which was treated. There was no history of a penile lesion or of injections for syphilis.

Physical examination disclosed a well-developed Negro walking in obvious pain. On both sides of the anus and directly opposed so as to make contact there were two areas of ulceration, each measuring 1.5 by 1.5 cm. The surfaces of these ulcerations were covered with raised red granulations. The underlying tissue felt nodular and indurated. There was no evidence of fissure, abscess or fistula. The sphincter tone was increased, and digital examination caused severe pain.

*From the Surgical Service, Lovell General Hospital, Fort Devens, Massachusetts.

†Junior assistant surgeon, Rectal Clinic, Boston Dispensary; assistant in rectal diseases, Brockton Hospital; formerly, surgeon in charge of rectal diseases, Lovell General Hospital, Fort Devens, Massachusetts.

The remainder of the examination was temporarily deferred, and it was suspected that the patient had an anal fissure.

Routine laboratory studies on admission were within normal limits. A Kahn test was negative. A routine Frei test was positive at 72 hours. The soldier was given hot sitz baths, with symptomatic relief. Further examination several days after admission was entirely within normal limits except for the indurated ulcerations. Sigmoidoscopy revealed no disease in the rectum or lower sigmoid. There was no evidence of anal fissure.

The ulcerations did not heal under palliative treatment, and the underlying induration persisted. On October 6, operation was performed. Minute abscess cavities were found beneath the ulcerations. On the left side of the anus these connected with each other and with a deep crypt at "9 o'clock" on the anorectal line. Fistulotomy was performed, with adequate shelving of the skin edges. The abscess cavities did not connect with the anal canal on the right side. These cavities were unroofed and packed with iodoform gauze. Sections of tissue were sent to the laboratory. Postoperative treatment consisted of hot sitz baths three times daily, a regular diet and mineral oil by mouth. Healing was slow.

The pathological report stated, in part, that the subepithelial layer was occupied by a large number of clear cells with vacuolated cytoplasm and small dark-staining nuclei (Fig. 1). Some of these cells contained coccoid or rodlike Donovan bodies. The diagnosis was granuloma inguinale.

The patient was placed on intramuscular Fuadin* therapy, receiving 1.5 cc. on the 1st day, 3.5 cc. on the 2nd day, 5.0 cc. on the 3rd day and 5.0 cc. every third day for six doses — a total of 40 cc. There was no reaction to the drug, and no pain resulted from the injections. The operative wounds closed steadily, leaving a soft healed scar with loss of normal skin pigment. Three weeks after the beginning of Fuadin therapy, the wounds were healed, and the patient was returned to duty.

FIGURE 1. *Case 1.*
Photomicrograph of the biopsy specimen shows marked hyperplasia of the epithelium. Characteristic large clear cells occupy the papillae and upper layers of the corium. Occasionally these cells contain Donovan bodies.

In the above case the patient was suspected of having an anal fissure because of the typical history of severe pain associated with bowel movements and lasting for one hour following them. The two areas of ulceration were thought to be due to chafing arising from intensive military training. They were discrete and directly apposed and were covered with pink granulations such as are found in all healing wounds. The positive Frei test was significant in that it indicated the coexistence of lymphogranuloma inguinale. It would have been impossible to make the correct diagnosis from the appearance of the lesion alone, and except for the pathological report it would have been entirely missed. Had the diagnosis been suspected, it might have been confirmed sooner by smear or by biopsy.

CASE 2. A 24-year-old Negro soldier was admitted to the Surgical Service of this hospital on October 25, 1944, complaining of anal pain and purulent anal discharge. In 1934, he first noticed a small papule on the right side of the anus. This papule caused no discomfort but slowly increased in size until it was as large as a walnut. In 1942, an abscess developed at the site of the mass, and this was followed by a fistula. Some type of surgery was performed at a civilian hospital, but apparently the growth was left undisturbed. The patient felt well until May, 1944, at which time a right perianal abscess recurred. This was incised and drained. Drainage ceased for a short period but recurred in the following September, when the patient was admitted to an Army hospital and transferred to the Lovell General Hospital for further treatment and disposition. He denied having had gonorrhea but stated that he was taking "injections for his blood."

Physical examination disclosed a well-developed Negro in no distress. There was a large granulomatous mass measuring 5 by 3 cm. just to the right of the anus. It extended medially to involve the anal canal and terminated laterally in two long, well-healed but indurated scars (Fig. 2). The center of the mass was ulcerated and covered with a purulent secretion, beneath which were easily bleeding pink granulations. Digital examination was negative except for hard indurated tissue of the right anal wall. The sphincter tone was slightly diminished. There were slight internal hemorrhoids.

FIGURE 2. *Case 2.*
Photograph of the granulomatous mass of ten years' duration.

Routine laboratory studies were within normal limits. A Kahn test was negative. Consultation was held with the Venereal Disease Section, with a resultant diagnosis of early latent syphilis. A Frei test was negative at 72 hours. On October 26, a biopsy specimen was taken under local anesthesia.

*Sodium antimony-III biscatechol disulphonate of sodium, manufactured by the Winthrop Chemical Company, New York City.

The pathological diagnosis was chronic suppurative, nonspecific inflammation. The degree of sphincter involvement could not be determined, but it was considered certain that extensive surgery would be required to eradicate the lesion.

The patient was returned to his unit on November 13 with a diagnosis of nonspecific granuloma, and it was requested that he be separated from the Service.

On December 14, a report was received from the Army Medical Museum on the biopsy material submitted to it; the diagnosis was granuloma inguinale. The patient was recalled at once to the hospital. He was complaining of a foul-smelling, purulent anal discharge. A biopsy was repeated. The resultant pathological report stated that here and there just beneath the epithelium there were marked edematous patches and a few large cells, with clear cytoplasm and round nuclei, some of which contained masses of rodlike or coccoid Donovan bodies deeply stained with blue (Fig. 3). The diagnosis was granuloma inguinale.

Intramuscular Fuadin therapy was instituted as in Case 1. After conclusion of the course, the patient was allowed a rest

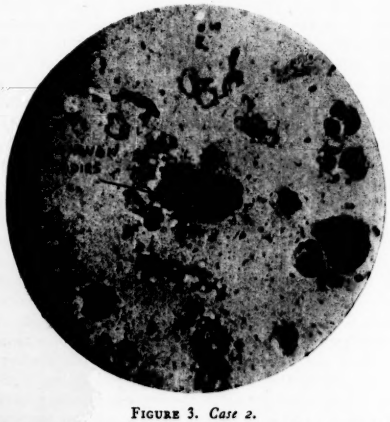


FIGURE 3. *Case 2.*

This photomicrograph of a stained smear shows numerous intracellular oval (Donovan) bodies in the cytoplasm of a large macrophage.

of 1 week. There was no reaction to the drug. On December 26, the patient stated that he was having much less drainage. The lesion appeared unchanged, but within the following week the granuloma decreased considerably in size and showed much less surrounding induration. Drainage had practically ceased, and there were no complaints. On January 15, 1945, a second course of Fuadin was begun. It was completed on January 24, and the patient was returned to duty. Subsequently the granuloma disappeared entirely, leaving a small area of skin in which the normal black pigment was absent (Fig. 4).

In this case granuloma inguinale had been present for approximately ten years, and the advanced granulomatous stage of the disease was well represented. Even though the diagnosis was suspected on admission, it was missed because of a poor section of tissue taken for biopsy. Two courses of Fuadin were given, a procedure that is recommended owing to the tendency to recurrence. Intramuscular Fuadin salvaged for military service a soldier who might otherwise have been lost to it.

SUMMARY

Granuloma inguinale is a chronic, progressive skin infection of the genitals, scrotum and inguinal regions. It occurs infrequently about the anus. Its etiologic agent is a gram-negative, nonmotile bacillus identifiable by smear or biopsy in the state of encapsulation as a "Donovan body." The disease occurs primarily in Negroes.

Two cases are presented involving the region about the anus, one in an early ulcerating stage and the other in an advanced granulomatous stage. The response to Fuadin was excellent in both cases but was especially dramatic in the second case, in which there was a lesion of ten years' duration.

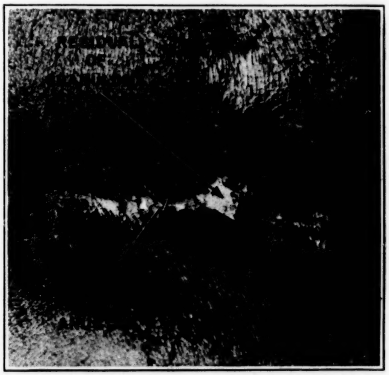


FIGURE 4. *Case 2.*

Photograph showing the healed lesion after two courses of Fuadin.

475 Commonwealth Avenue

BIBLIOGRAPHY

Anderson, K., De Monbreun, W. A., and Goodpasture, E. W. Etiologic consideration of Donovania granulomatis cultivated from granuloma inguinale (3 cases) in embryonic yolk. *J. Exper. Med.* 81:25-40, 1945.

Anderson, K., Goodpasture, E. W., and De Monbreun, W. A. Immunologic relationship of Donovania granulomatis to granuloma inguinale. *J. Exper. Med.* 81:41-50, 1945.

Brandt, R., and Gatewood, T. S. Early diagnosis of granuloma inguinale. *Am. J. Syph., Gonor. & Ven. Dis.* 25:48-55, 1941.

Kolmer, J. A. *Clinical Diagnosis by Laboratory Examination.* 1239 pp. New York: D. Appleton-Century Company, 1944.

Kornblith, B. A. Intradermal reaction as aid in diagnosis of granuloma inguinale. *New York State J. Med.* 44:2476-2478, 1944.

Robinson, H. M., Robinson, H. M., Jr., Shelley, H. S., and Mays, H. B. Treatment of granuloma inguinale. *South. M. J.* 35:889-895, 1942.

Shelley, H. S. Surgical treatment of granuloma inguinale. *Bull. School Med. Univ. Maryland* 25:105-108, 1940.

MEDICAL PROGRESS

RESEARCH IN PHYSICAL MEDICINE (Concluded)

ARTHUR L. WATKINS, M.D.*

BOSTON

ELECTROTHERAPY

Diathermy

Medical diathermy has been defined as the therapeutic use of heat generated in body tissues by a high-frequency current that has insufficient local intensity to produce temperatures high enough to destroy or injure tissues. These currents are applied locally by three methods: conventional long-wave diathermy with contact metal electrodes; short-wave diathermy with an electric field, air space or insulated electrodes being used; and short-wave diathermy with an electromagnetic field, a cable or coil being employed. Conventional or long-wave diathermy frequency is between 500,000 and 3,000,000 cycles per second, whereas short-wave diathermy frequencies are between 10,000,000 and 100,000,000 cycles. At the present time short-wave diathermy is used much more extensively than is long-wave diathermy, because of its ease of application, the somewhat less danger of burns and the more uniform temperature effects. The fundamental laws and concepts of alternating current phenomena on which short-wave diathermy technic is based have been well described in Osborne and Holmquest's[36] textbook on electrotherapy. A shorter summary has recently been written by Holmquest.[37] In summary, he states that in practice there are two methods of applying high-frequency power to tissue for treatment purposes — by means of the high-frequency field such as exists between plate electrodes, with or without an air space between electrodes and skin, and by means of the high-frequency magnetic field that is set up by the high-frequency current flowing through a coil, which is wound around the part to be treated or wound into a circular pad and placed over the tissues in which it is desired to generate heat.

Much investigative work has been done to determine the heating characteristics of these two methods. It has been shown that, with the high-frequency electric field, heating depends on the specific conductivity of the electrolyte exposed to the field and rises to a maximum for a certain conductivity. As the frequency of the field is increased, the conductivity at which maximal heating occurs also increases. It has been shown that there is a linear relation between frequency and specific conductivity for maximal heating. With a frequency of 10 megacycles (wave lengths of 30 meters), maximal heating occurs in a sodium chloride solution having a specific conductivity of 5.4 for a concentration of 0.0045 gm.-mols per liter or 0.0263 gm. per 100 cc. of solution. Since for dilute solutions conductivity is proportional to concentration, one can readily determine the approximate concentration of sodium chloride solution into which the rate of heat production would be graded for various frequencies. The concentration of sodium chloride having the conductivity of blood plasma is 0.85 gm. per 100 cc. of solution. So that heat shall be produced predominantly in the vascular type of tissue where it is normally produced through energy metabolism, an extremely high frequency must be employed. It must not be inferred, however, that, the approximate specific conductivity of various organs of the body being known, frequencies can be selected to elevate the temperature of these organs without elevating the temperatures of other tissues and organs. Efficiency of the blood stream in dissipating heat and conveying it to other structures of the body tends to equalize temperatures and hence to prevent such specific heating.

Regarding the second method of applying high-frequency energy to the tissues, experiments have shown that the induction field generates heat in an electrolyte in direct proportion to the conductivity of the electrolyte. Such a field should produce heat dominantly in vascular tissue. Actual measurement of temperatures obtained in the deep tissues of living subjects has confirmed this theoretical deduction. From time to time claims are made for specific biologic and bactericidal effects of various frequencies. Careful investigators seem agreed, however, that the only demonstrable effects of short-wave diathermy are a production of heat and the physiologic responses that normally follow production of heat in tissue. Hence, the method of applying short-wave diathermy that develops heat primarily where oxidation produces it naturally in the human body, thereby inducing and maintaining active hyperemia and permitting the warmed circulating blood to convey heat to other tissues, seems to be the preferable manner of treatment of all conditions that might be benefited by such therapy.

The technic of application of short-wave diathermy has been well described in the standard textbooks.

*Associate in medicine, Harvard Medical School; chief of physical medicine, Massachusetts General Hospital.

Regulation of dosage, however, is still empirical. It is necessary to rely on a comfortable heat sensation of the patient's skin in measuring doses, since the milliammeter on the apparatus does not measure the energy passing through the patient. Some experimental work has been done on the measurement of field strength, but these methods have not as yet been applied clinically.[38] Until further evidence is forthcoming, it is sufficient to say that the physiologic effect of diathermy is simply that of production of heat in the tissues.

Electrodiagnosis

The chief diagnostic function of specialists in physical medicine is appraisal of excitability of nerve and muscle by stimulation with appropriate electrical current. In considering the methods used for this purpose, the average physician or surgeon has been cognizant only of the test for the so-called "reaction of degeneration," as described by Erb. Recent developments in electrical engineering have made available for experimental and clinical use new instruments capable of producing currents of easily measured strength, duration and frequency of impulse. A number of laboratory and clinical studies based on these new methods have been published in the last few years. They are of particular interest at this time because of the frequent occurrence of peripheral-nerve injuries among war casualties.

In Erb's test, faradic and galvanic currents are used, and the preservation of normal responses to these currents serves to distinguish severe peripheral-nerve injuries from minor lesions, upper-motor-neuron disease and hysterical paralysis. The limitations of this test are, however, frequently overlooked. Of first importance is the fact that changes in the electrical reactions of paralyzed muscles do not appear, even after complete severance of the motor-nerve supply, before a minimum of four to five days and may not be completely developed for two weeks. Electrical tests are therefore of no diagnostic value unless time is allowed for these changes to occur. The responses of muscles that when tested for a sufficient length of time after injury indicate loss of conduction of the normal nerve impulse and degeneration of intramuscular nerve fibers have been described by Erb as the reaction of degeneration. Recently Pollock et al.[39] reinvestigated Erb's conclusions, making use of modern apparatus, and found that his description was essentially accurate. Denervated muscles do fail to respond to a faradic current when stimulated percutaneously. This is not absolutely true, however, for even completely denervated muscles respond to the faradic current if a sufficiently high voltage is used. It should be further pointed out that although only the faradic currents were used by Erb for this test it is more usual at present to employ a 60-cycle alternating, sinusoidal current, since it is of proper frequency to produce a tetanic contraction in normal muscle and the separate impulses are of sufficiently short duration so that the denervated muscle does not respond with the voltage of current usually applied clinically. Consequently, a more accurate description of such a reaction would be to say that there is loss of response to a tetanizing current.

The type of response to stimulation by direct or galvanic current is of equal or even greater importance. Normal muscles respond to a direct-current impulse by a single quick twitch and rapid relaxation. Denervated muscles, on the other hand, with the same type of stimulus show a slow, wave-like contraction, which is frequently sustained to a lesser degree without complete relaxation so long as the current is applied. Pollock et al. describe this response as a tetanic contraction and emphasize its importance in following the state of innervation of muscle during various stages of degeneration and regeneration. Doupe[40] studied this response of denervated muscle to direct-current stimulation by means of simultaneous mechanical and action-potential recordings. He found that the sustained contraction was explained by repetitive excitation and described the phenomenon as galvanotonous, which is perhaps a better description than Pollock's, since clinically the contraction is not so great as that of a complete tetanus. Doupe also found that the temperature of the denervated muscle was below normal and that the slowness of contraction could be partially explained on this basis, since the prolonged excitation factor was also accentuated by cold, as was a hyperirritability to currents of long duration and threshold strength.

In addition to the sluggishness of contraction on galvanic stimulation, Erb mentioned a change in the response to positive and negative currents. Whereas the cathode-closing contracture is normally greater than the anode-closing contracture, during denervation the responses may be equal or occasionally reversed. Pollock et al.[39] and other observers[41] have found that the threshold response to both cathode and anode stimuli may become equal during denervation but have never seen true reversal. Pollock has emphasized another feature of the degenerative reaction to direct-current stimulation — namely, hyperirritability. This may be more apparent than real, however, since the muscle contraction is increased because of prolonged excitation.

In addition to stimulating muscles directly with galvanic or tetanizing (faradic) current, it is also advisable to test conduction of nerve impulse by stimulating the nerve directly whenever possible, preferably above the site of the lesion. For this purpose either galvanic or faradic current may be used. The degenerated nerve does not transmit either current, and in this case responses are obtained only when the muscle is stimulated directly. Preservation of conduction along the nerve and of normal

faradic response when the testing is done sufficiently long after injury is accordingly of definite value in separating severe from minor injuries.

These electrical tests described are, however, of little value in determining the first signs of recovery or of nerve regeneration, since it is generally accepted that voluntary return of function precedes return of faradic response. For accurate quantitative study of electrical excitability of nerves and muscles, the factors of current strength, duration and rate of increase of potential must be considered. Electrical generators are now available that enable one to control and measure stimulation. It has been found that when the development of current is essentially instantaneous, as with condensor discharges or square-wave impulses, there is a definite relation between the strength and the duration of impulse required to induce a barely perceptible standard or threshold contraction of the muscle. By stimulating muscles with currents of assorted strengths and durations, it is possible to plot the responses in the terms of a strength-duration curve that gives a graphic representation of the electrical excitability. Repeated determinations of such curves give fairly accurate qualitative measures of excitability, and at times changes may be detected in these curves that indicate beginning regeneration before it can be detected clinically.[42, 43] Such a change may be either a shift in the total curve toward normality or the presence of a break in the curve, indicating fibers of different excitability, owing to the arrival in the muscle of some regenerated nerve fibers.[44]

An arbitrary point on the strength-duration excitability curve has been designated as the chronaxie, or time factor of excitability. The first step in chronaximetry is to measure the voltage of current necessary to cause a liminal response when a unidirectional current of long duration is applied. This measurement is called the rheobase. The length of time that a current of twice the rheobase voltage must be applied to produce the same response is the chronaxie. Normally the chronaxie is extremely short, — 0.001 second or less, — but with denervation it becomes ten or even more than a hundred times greater. Chronaxie measurements are of greater quantitative value than are galvanic and faradic tests, but they do not offer so complete information as does determination of the whole strength-duration curve and are subject to more possibilities of error in the single determination as compared with the many points on the strength-duration curve.

Currents of various wave forms have also been employed to stimulate muscles for electrical diagnosis.[45] These may be in the shape of triangles, with a gradual increase of current to a peak and a similar decline, or progressive currents, or the wave form may be that of a true sine wave. The currents are used to measure another factor in electrical excitability — namely, the power to accommodate to a slow change in potential. Because of this power, normal muscle does not respond to currents with a slow increase of potential except at high-current value. Denervated muscles, however, lose this characteristic and consequently respond to progressive currents of long duration and to extremely slow sinusoidal currents of low voltage. This change in accommodation, as indicated by the responses to progressive currents of long duration or to sinusoidal currents of low frequency, coincides with the changes in other measurements of excitability previously mentioned. Generators capable of producing progressive currents and low-frequency sine waves are to be found only in experimental laboratories, and consequently these tests do not have a wide clinical use.

All measurements of electrical excitability must be interpreted in proper relation to the time elapsed since injury or surgery. First, as previously mentioned, one or two weeks must elapse for the possible appearance of degeneration. Later, when one looks for regeneration, the maximum speed of recovery, approximately 4 millimeters a day, must be considered. Local factors, such as atrophy of the skin, edema and ischemia, may cause variations in the results. Perhaps most important of all is the skill, experience and diligence of the investigator in developing a standardized technic so that the results of one examination may be compared with those of another. The most valuable and accurate results are obtained by using a battery of electrodiagnostic tests, which are then repeated at stated intervals to determine the state of progress.

A new and entirely different method of electrodiagnosis, known as electromyography, makes use of the recording of small electrical potentials from muscles at rest or during voluntary contraction. Since these potentials are of extremely low voltage and short duration, a highly sensitive amplifier and recording system, such as the cathode-ray oscilloscope or amplifiers employed for recording brain potentials, must be employed. Bipolar electrodes are used, either percutaneously or by inserting a concentric needle into the muscle to be investigated. With these technics normal resting muscles give rise to no electrical discharges. During voluntary contraction, action potentials of a relatively high frequency and voltage and characteristic appearance are recorded.[46] These action potentials are, of course, not obtained in paralyzed muscles with complete denervation. Partially denervated muscles, on the other hand, may discharge scattered diphasic spikes, indicating some motor-unit activity, although no visible contraction is observable. The appearance of these spikes accordingly quickly differentiates a complete from a partial lesion.

In the case of complete degeneration, no action potentials are recordable, but if a proper type of amplifier is used, such as a cathode-ray oscillograph,

tiny spontaneous resting discharges of low voltage and duration may be recorded and identified as fibrillation of denervation, giving positive evidence of a complete lesion.[47]

Even with this sensitive type of instrument, there is a necessary long period of waiting before the slow process of nerve regeneration can be detected peripherally in the muscle. Electromyography is, however, of considerable value in picking up the first signs of neurotization of muscle in the form of spontaneous discharges of high voltage, indicating beginning motor-unit activity.

Another electrical aid in the measurement of nerve function is determination of skin resistance. For this purpose Richter and Katz[48] have described a simple dermometer suitable for quickly mapping areas of high resistance to the passage of a direct current. These areas correspond to the distribution of sensory innervation of peripheral nerves, particularly the ulnar and median. The obvious advantage of such a test is that it is completely objective and does not require the co-operation of the patient. It is especially useful in cases of suspected malingering or hysteria. In my experience, the regeneration of sensory nerves can be detected much earlier by the usual clinical test of sensation than by measurement of the skin resistance. In some cases there is a lag of several months after sensation has returned before the skin resistance decreases to normal values.

Electrotherapy for Denervated Muscles

The value of electrical stimulation of a denervated muscle has frequently been debated in the literature.[49–52] It is generally conceded, however, that stimulation of muscles during the first few weeks following denervation does not prevent the occurrence of denervation atrophy. The results in experiments that were continued for longer periods of time indicate that the rate of atrophy in the third and fourth weeks and later is considerably delayed by appropriate electrical stimulation of the denervated muscle.[53] It has also been shown that although the rate of regeneration of the nerve is not affected by this treatment the return of voluntary muscle contraction is enhanced by electrotherapy and that the degree of fibrosis is lessened according to histologic examination. In the light of more recent studies, the negative effects of galvanic stimulation of denervated muscle have been explained as due to ineffectual or too infrequent stimulation. Daily treatment has been found to be more efficient than treatment on alternate days, and for optimum effect a stimulus should be chosen that will produce a maximal tension during contraction.[54]

In view of the latest experimental work, it appears that the ordinary type of clinical muscle-stimulating apparatus is inadequate to achieve optimum therapeutic results. The rate of rise of each impulse should be slow, and the frequency of impulses also should be slow, in the neighborhood of 10 to 25 per second, in order to cause maximum contraction of the denervated muscle with a minimum of current strength.[55]

Recent experiments have also shown that the dangers of fatiguing muscles have been overemphasized in the past. Marked fatigue through artificial stimulation or forced exercise was found to have no deleterious effect on regeneration. It now seems apparent that maximum results in strengthening a muscle can be obtained only by producing a maximal degree of tension.[54]

Ion Transfer (Iontophoresis)

Direct current may be used to drive certain medicinal electrolytes into tissues by the force of the polar reactions. The depth of penetration, however, is limited because of the far higher velocity and greater number of the tissue ions. The foreign ions introduced through the skin lose their electrical charge and are precipitated as soluble or insoluble compounds in the superficial tissues. The speed of circulation also serves to dilute the new ions quickly. In spite of these limitations, definite local and systemic effects can be produced by this method.

The drugs most widely used for the vasodilatory effect are histamine and mecholyl (acetylbeta-methylcholine chloride). There are a great many clinical reports in the literature describing the use of these and other drugs in ion transfer. These have been summarized by Osborne.[56] Very little fundamental research has been done, however. More work like that of Pereyra[57] is needed to clarify the subject. He studied microscopically the penetration of copper by iontophoresis in experimental animals, and found that the greatest depth effects were obtained when a solution such as aerosol MA was added to inhibit the binding of copper by tissue proteins. Quantitative studies by Molitor and Fernandez[58] provide data for safe dosage.

Hydrotherapy

The use of water for therapeutic purposes is well established. Certain physical characteristics are recognized to be of importance. Water is an extremely flexible therapeutic agent, being easily changed from a liquid to a gaseous or solid state within relatively narrow temperature ranges, which may be easily measured. It is an excellent heating or cooling medium because of its high conductivity. Since it has a high specific heat, large amounts of heat are necessary to increase its temperature, and hence it is an excellent cooling agent. Similarly, it liberates a large amount of heat when cooled, making it a good heating agent. The latent heat of vaporization is high, and the heat lost in insensible perspiration is consequently significant in the regulation of the body temperature. The evaporation of sweat accounts for the ability to lose heat rapidly. The

buoyant effect of water during immersion is taken advantage of in forms of muscle re-education with underwater exercises.

The mechanical impact of water is also known to produce physiologic responses. The type of impact may be varied, as with showers, douches, bubbles and so forth, and sedative and stimulative effects can be observed clinically. The exact mechanism of these reactions has never been extensively investigated and is a field of research in physical medicine that needs to be further developed. The best known effects of water are those that are dependent on its temperature, and these have been discussed previously.

Mechanotherapy

Massage

Knowledge of the physiologic effects of massage is rather limited. In actual practice, massage is usually preceded by application of heat and frequently followed by exercise. Improvement in circulation, relief of muscle spasm and an increase in joint range and strength of movement may be readily observed clinically. The exact role of massage in securing these effects has received little investigative study. Recent reviews on the physiology of massage[59, 60] refer to studies done for the most part twenty to twenty-five years ago. Papers published since then have been almost exclusively clinical in nature, with the emphasis on technic. The early studies suggested that the twenty-four-hour excretion of nitrogen was increased on days that massage was administered. Diuresis and an increase in oxygen consumption and carbon dioxide production following general or abdominal massage have also been observed. More recent workers have failed to find any significant alteration of the metabolism in the normal subject, and at the present time it is generally believed that whatever metabolism changes may be produced by massage are secondary to the mechanical influence on the circulation of the parts concerned.

The effects of massage on circulation are more definite and are easy to demonstrate. Light stroking has been shown to produce an almost instantaneous, although transient, dilatation of the capillary vessels, with a more enduring effect when the pressure is heavier.[61] Photographic studies of capillary circulation have also indicated changes in vessel walls that permit the passage of leukocytes. Some observers have found that the number of circulating red cells may also be increased by massage, although the exact mechanism is not explained.[62] It is well known that lymphatic circulation is increased by massage and passive motions.[63, 64]

It is inferred by clinical observation that massage affects the nervous system. Part of the dilatation of blood vessels is thought to be effected through sympathetic reflexes. Massage is probably most frequently employed for relief of pain resulting from muscle spasm. Massage is also used for its general sedative effect and for its hypnogogic action. Massage is also said to have stimulative effects on the neuromuscular apparatus. It is certainly an adequate stimulus for exciting tendon reflexes in spastic patients. There is, however, an absence of studies that explain the mechanism of such effects, which are thought to be achieved through the action of massage on the nerves and muscles.

The practice of massage at present seems to be more of an art than a science. Physicians and technicians through years of experience may learn how to achieve the desired results, often by an individual technic. Prescription of massage consequently amounts to instructing the technician in the results desired, whether stimulative or sedative, and including instruction in the type of manipulation to be avoided. Considering that this method of treatment has been observed clinically to produce important beneficial effects in a large variety of conditions, it is unfortunate that more scientific knowledge concerning the mechanisms of its actions is not available.

Exercise

The subject of exercise has many ramifications into the fields of mechanics, biochemistry and physiology in health and disease. Attention is called to several recent reviews. Elkins[65] has emphasized that those prescribing therapeutic exercise should have a knowledge of the basic principles involved in muscular action. One of the primary factors relates to the mechanics of movement. The bones and joints make up a fairly intricate lever system, with changing forces depending on the angle of pull. Two other physical properties of muscle of considerable significance are its elasticity and its extensibility. Contractility of muscle varies with its length, and this has to be taken into consideration in performing re-education exercises. The control of muscle movement through the nervous system includes resting and postural tonus, as well as co-ordination and skillful activity. These topics have been briefly reviewed by Elkins, who also cites further references.

An aspect of locomotion of considerable importance is that of posture or body mechanics. From the point of view of the physician, there is a strong feeling, based on clinical evidence, that good posture is essential for health and that poor body mechanics may be responsible for a number of pathologic conditions. This point of view has been stressed for a number of years by Goldthwait, Brown, Swaim and Kuhns[66] and was recently summarized by Hansson.[67] That stresses and strains on joints and supporting structures may result from poor mechanics is fairly obvious. Physiologic studies on the effects of posture on the various systems of the body, however, have not produced evidence in defense of the medical opinions of the relation be-

tween posture and disease, except in some extreme cases, such as scoliosis with compression on the thorax. Hellebrandt and Franseen[68] have shown that compensatory mechanisms are developed in man that cancel the apparent mechanical disadvantage of the vertical stance in overcoming gravitational stresses. Standing has been found to be cheap in terms of metabolic cost, and it has yet to be unequivocally demonstrated that improvement in body mechanics is associated with a significant decrease in exchange of energy.[69] The circulatory system is probably most affected by posture. The blood pressure decreases with the erect position, but there are many compensatory mechanisms that act to overcome the disparity between the size of the vascular bed during standing and the volume of flow. Involuntary postural sway may be of considerable importance in stimulating reflex activities of vital centers located in the medulla, making circulatory adaptation more efficient. Further studies of these compensatory mechanisms in health and disease are necessary to put the concept of what is correct posture on firm scientific ground.

Another aspect of exercise that has lately aroused considerable interest is the problem of general physical fitness. Much of the recent work is subject to rules of military secrecy and has therefore not yet been published. Physical fitness has frequently been assessed by exercise tests, such as the step test or treadmills and bicycle ergometers. During such periods of exercise observations are made of the circulation and respiration and of various aspects of blood chemistry. Over a hundred papers on this subject have recently been reviewed by Taylor.[70] Physical training has been found to increase exercise performance. The physiologic results of training indicate that except for a small but consistent reduction in heart rate, changes in resting functions are either nonexistent or slight. In submaximal work, mechanical efficiency is increased by training and physicochemical blood factors depart less from their resting values. Coincident with substantial training, increments in maximal work output, maximal blood lactate, oxygen consumption and oxygen depth undergo increase. Improvement in athletic type-fitness tests amounts to as much as 20 per cent. Absolute muscle power has been shown to rise 30 per cent with training but is indefinitely maintained in the post-training period, whereas endurance depreciates rapidly to 50 per cent.[71]

The problem of fatigue has stimulated considerable investigative work, including study of mental and emotional variables. Neurocirculatory asthenia, the so-called "effort syndrome," is generally believed to be a psychoneurotic manifestation. The general problem of muscular exercise and fatigue in disease has been well reviewed by Simonson and Enzer.[72] It is to be expected that the final analysis of the large body of new information on exercise and fatigue that is being developed in relation to the recent war will place exercise therapy on a firm scientific basis.

Occupational Therapy

The medical profession in general is little acquainted with the potential value of occupational therapy. Those clinically familiar with it have realized that muscular strength, co-ordination and adaptability of injured or diseased parts may be gained under the skilled direction of an occupational therapist. It has been emphasized that mental as well as physical function is stimulated by such activities. Psychiatrists have found occupational therapy to be an essential part of a therapeutic program, particularly for hospitalized patients. Occupational therapy may also take the form of prevocational training as a part of adequate rehabilitation of injured persons.

The medical literature, however, is extremely deficient in case studies and progress reports on occupational therapy. Basic research, consisting of kinesiologic studies and investigation of muscular-skeletal pathologic conditions, is also inadequate. This sort of research is needed, together with studies aimed at evaluating certain crafts and shopwork activities, with the object of analyzing the motions required and their adaptability to applied kinesiology. Familiarity with the present knowledge of technics and their effects should lead to further employment of this therapeutic agent and stimulate necessary studies.

References

36. Osborne, S. L., and Holmquest, H. J. *Technic of Electrotherapy and Its Physical and Physiological Basis.* 760 pp. Springfield, Illinois: Charles C Thomas, 1944.
37. Holmquest, H. J. Physical therapy: low- and high-frequency fields and currents. *M. Physics.* Pp. 1074-1093, 1944.
38. Lion, K. S. Dosimetry and local distribution of energy in electric high frequency field. *J. App. Physiol.* 14:545-551, 1943.
39. Pollock, L. J., et al. Reaction of degeneration in electrodiagnosis of experimental peripheral nerve lesions. *War Med.* 7:275-283, 1945.
40. Doupe, J. Studies in denervation: contractility and excitability of denervated muscle. *J. Neurol. & Psychiat.* 6:141-153, 1943.
41. Huddleston, O. L. Experimental studies on electrical reactions of denervated skeletal muscles. *Arch. Phys. Med.* 26:197-205, 1945.
42. Watkins, A. L. Electrical aids in diagnosis and prognosis of nerve injuries. *Arch. Phys. Therapy* 23:76-83, 1942.
43. Ritchie, A. E. Electrical diagnosis of peripheral nerve injury. *Brain* 67:314-330, 1944.
44. Pollock, L. J., Golseth, J. G., and Arieff, A. J. Use of discontinuity of strength duration curves in muscle in diagnosis of peripheral nerve lesions. *Surg., Gynec., & Obst.* 79:133-141, 1944.
45. Pollock, L. J., Golseth, J. G., Arieff, A. J., and Mayfield, F. Electrodiagnosis by means of progressive currents of long duration. *Surg., Gynec. & Obst.* 81:192-200, 1945.
46. Watkins, A. L., and Brazier, M. A. B. Studies on muscle innervation in poliomyelitis and nerve injuries. *Arch. Phys. Med.* 26:69-75, 1945.
47. Weddell, G., Feinstein, B., and Pattle, R. E. Electrical activity of voluntary muscle in man under normal and pathological conditions. *Brain* 67:178-257, 1944.
48. Richter, C. P., and Katz, D. T. Peripheral nerve injuries determined by electrical skin resistance method: ulnar nerve. *J. A. M. A.* 122:648-651, 1943.
49. Chor, H., Cleveland, D., Davenport, H. A., Dolkart, R. E., and Beard, G. Atrophy and regeneration of gastrocnemius-soleus muscles. *J. A. M. A.* 113:1029-1033, 1939.
50. Fischer, E. Effect of faradic and galvanic stimulation upon course of atrophy in denervated skeletal muscles. *Am. J. Physiol.* 127: 605-619, 1939.
51. Hines, H. M., Thomson, J. D., and Lazere, B. Physiologic basis for treatment of paralyzed muscle. *Arch. Phys. Therapy* 24:69-73, 1943.
52. Jackson, E. C. S., and Seddon, H. J. Influence of galvanic stimulation on muscle atrophy resulting from denervation. *Brit. M. J.* 2: 485,1945.
53. Gutmann, E., and Guttmann, L. Effects of electrotherapy on denervated muscles in rabbits. *Lancet* 1:169, 1942.

54. Wehrmacher, W. H., Thomas, J. D., and Hines, H. M. Effects of electrical stimulation on denervated skeletal muscle. *Arch. Phys. Med.* **26**:261-266, 1945.
55. Grodins, F. S., Osborne, S. L., Johnson, F. R., and Ivy, A. C. Stimulation of denervated skeletal muscle with alternating current. *Am. J. Physiol.* **142**:216-221, 1944.
56. Osborne, S. L. Direct current applications and electrical stimulation of muscle. A chapter in *Handbook of Physical Medicine*. 392 pp. Chicago: American Medical Association, 1945. Pp. 192-201.
57. Pereyra, A. J. Penetration of surface tissues with copper by iontophoresis: penetration with organic and inorganic copper salts and use of detergents in iontophoresis. *Arch. Dermat. & Syph.* **52**: 96-105, 1945.
58. Molitor, H., and Fernandez, L. Studies on iontophoresis: experimental studies on causes and prevention of iontophoretic burns. *Am. J. M. Sc.* **198**:778-785, 1939.
59. Pemberton, R. Physiology of massage. A chapter in *Handbook of Physical Medicine*. 392 pp. Chicago: American Medical Association, 1945. Pp. 69-83.
60. Scull, C. W. Massage: physiologic basis. *Arch. Phys. Med.* **26**: 159-167, 1945.
61. Carrier, E. B. Studies on physiology of capillaries: reaction of human skin capillaries to drugs and other stimuli. *Am. J. Physiol.* **61**: 528-547, 1922.
62. Schneider, E. C., and Havens, L. C. Changes in blood after muscular activity and during training. *Am. J. Physiol.* **36**:239-259, 1915.
63. Bauer, W., Short, C. L., and Bennett, G. A. Manner of removal of proteins from normal joints. *J. Exper. Med.* **57**:419-433, 1933.
64. Drinker, C. K., and Yoffey, J. M. *Lymphatics, Lymph, and Lymphoid Tissue: Their physiological and clinical significance*. 406 pp. Cambridge: Harvard University Press, 1941. P. 115.
65. Elkins, E. C. Some principles which underlie therapeutic exercise. *M. Clin. North America* **27**:923-950, 1943.
66. Goldthwait, J. E., Brown, L. T., Swaim, L. T., and Kuhns, J. G. *Essentials of Body Mechanics in Health and Disease*. Fourth edition. 337 pp. Philadelphia: J. B. Lippincott Company, 1945.
67. Hansson, K. G. Body mechanics and posture. *J. A. M. A.* **128**: 947-953, 1945.
68. Hellebrandt, F. A., and Franseen, E. B. Physiological study of vertical stance of man. *Physiol. Rev.* **23**:220-255, 1943.
69. Hellebrandt, F. A., Brogdon, E., and Tepper, R. H. Posture and its cost. *Am. J. Physiol.* **129**:773-781, 1940.
70. Taylor, C. L. Exercise. *Ann. Rev. Physiol.* **7**:599-622, 1945.
71. Maison, G., and Kotalik, G. Further investigation of training pattern of human muscle. *Fed. Proc.* **1**:56, 1942.
72. Simonson, E., and Enzer, N. Physiology of muscular exercise and fatigue in disease. *Medicine* **21**:345-419, 1942.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

BENJAMIN CASTLEMAN, M.D., *Associate Editor*

EDITH E. PARRIS, *Assistant Editor*

CASE 32191

PRESENTATION OF CASE

A seventeen-year-old schoolgirl entered the hospital because of fever and swelling of the ankles.

At the age of three years, following an attack of acute appendicitis, the patient developed migratory polyarthritis, a heart murmur and chorea. She was kept in bed for six months, then allowed up on slightly restricted activity. When she was seven years old there was a second bout of migratory polyarthritis, of unknown duration, without recurrence of chorea. There were no cardiovascular symptoms. After the second attack she continued in good health until six months before admission, when she twisted her right leg while swimming. Soon afterward the right leg became painful from the knee down. A chiropractor manipulated the leg and the pain disappeared. A week or two later she caught a "head cold," without a sore throat but associated with a temperature of 100°F. and a two-day bout of sharp pain in the left upper quadrant of the abdomen. One month later (five months before admission) she was admitted to a hospital with fever that was immediately relieved by sulfadiazine and penicillin. Two months before admission she was sick again, this time at home in bed with fever, muscle aches, cough and swollen ankles. After three weeks she was able to walk about but continued to have fever and ankle edema, which disappeared with rest. The night before admission she developed nausea, vomiting and a "stomach-ache," which persisted until she entered the hospital.

On physical examination there was a patchy erythema over the abdomen. The left border of the heart was 12 cm. to the left of the midline, with the maximal impulse in the fifth interspace. There was an apical systolic thrill. The mitral first sound was forceful, followed by a blowing Grade IV systolic murmur. The pulmonic second sound was greater than the aortic. The lungs were clear. The tip of the spleen was palpable and nontender, but the liver was not felt. There were two punctate, red, nontender areas on the cushion and tip of the left thumb.

The temperature was 99.6°F., the pulse 94, and the respirations 18. The blood pressure was 130 systolic, 80 diastolic.

The red-cell count was 3,470,000, with 8.2 gm. of hemoglobin. The white-cell count was 21,700, with 82 per cent neutrophils. There was a slight trace of albumin in the urine, and the sediment contained 2 red cells and 25 white cells per high-power field. In the chest film the heart appeared normal except for enlargement of the left auricle posteriorly, best demonstrated fluoroscopically. The electrocardiogram was normal. The sedimentation rate was 1.45 mm. per minute (normal, less than 0.35 mm.).

During the first three hospital days the temperature steadily rose to 104°F. and then spiked to 103 or 104 daily for a week. Blood cultures on the second and third days were negative, but five out of eight taken during the hospital stay were positive for an alpha-hemolytic streptococcus of the Lancefield Group A type. With the rise in fever an unproductive cough came on, which was accompanied by a "stitch" in the left side and was sometimes intensified by inspiration.

By the fourth day a ground-glass density had appeared in the chest film, which obliterated the left half of the diaphragm and the left costophrenic

sinus, as well as the left border of the heart and lower half of the left chest. The heart was displaced to the left (Fig. 1). The cardiac pulsations were vigorous. The spleen was not palpable. A few alpha-hemolytic streptococci grew in the throat culture. On the ninth day a macular rash appeared on the arms and backs of the hands and knuckles. On the thirteenth day the temperature peak was 100°F., and it was never any higher from that time on. Signs of fluid in the left chest gradually disappeared. There was no fever from the fourteenth to the eighteenth day. About the eighteenth day she began to complain of a dull constant ache in the muscles of the right thigh anteriorly, interrupted by occasional sharp pains in that region. On the twentieth day she complained of nausea, anorexia and periumbilical discomfort. The blood penicillin level on the twenty-fourth day was reported to be at least 0.06 but less than 0.24 units per cubic centimeter. The organism was inhibited in vitro by 0.06 units of penicillin per cubic centimeter. On the thirty-third hospital day the patient noticed aching pain in the left epigastrium and midepigastrium. It was similar to the attack four weeks previously, being constant and questionably aggravated by respiration. There was mild tenderness in the left upper quadrant. Dullness again appeared over the left chest posteriorly. The white-cell count was 20,000, with 73 per cent neutrophils, 13 per cent lymphocytes and 14 per cent monocytes. The legs and thighs were normal. The temperature and pulse did not change.

On the following evening the patient suddenly clutched the left upper abdomen, vomited and began writhing and groaning. She was pale, cold and sweating. The breath sounds were equally good on both sides, but dullness persisted at the left base. The trachea was not displaced. The heart sounds were rapid and regular, and the murmurs unchanged. The abdomen was quite silent, but at one time fairly normal peristalsis was heard. The abdomen was negative for fluid, spasm and tenderness. The knee jerks were hypoactive but equal. The patient was using both arms and both legs equally well in twisting and turning. She responded to questions by grunting. Oxygen slightly improved the color of the skin. Death occurred thirty minutes after the onset of this acute episode.

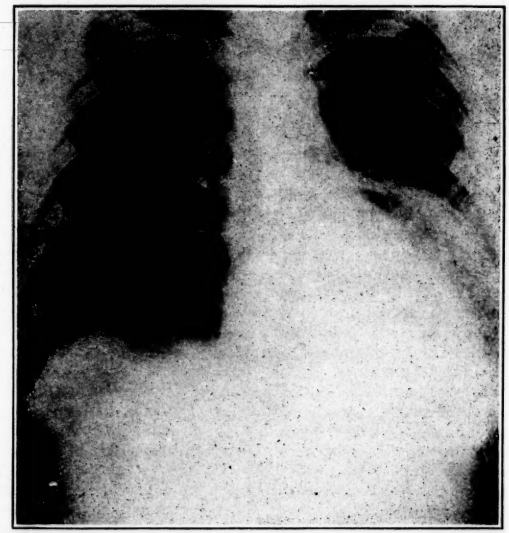


FIGURE 1. *Roentgenogram of Lung.*

DIFFERENTIAL DIAGNOSIS

DR. ALFRED KRANES: There can be little doubt that this patient was suffering from subacute bacterial endocarditis, and I find it very difficult to

consider seriously any other disease in the differential diagnosis. We shall start the discussion then by assuming that this patient had subacute bacterial endocarditis. Certainly the presence of long-continued fever, anemia, muscle and joint pains and splenomegaly — at least I assume that the spleen was enlarged, although it was felt only on admission — occurring in a patient whose heart was previously damaged by two attacks of rheumatic fever is a reasonable background for assuming that the patient had bacterial endocarditis. When, in addition, five blood cultures are positive for alpha-hemolytic streptococci the diagnosis seems well-nigh conclusive. There are, to be sure, several findings frequently found in the disease that were not encountered here, but their absence argues little, if any, against it. No mention is made of clubbing of the fingers or of petechiae, unless one assumes that the two punctate hemorrhages on the left thumb were petechiae.

The disease probably began five or six months before admission, when she first complained of pain in the right lower leg, although that, of course, may have been a mechanical strain from swimming. On the other hand, people with bacterial endocarditis frequently have bizarre muscle and joint pains. The former are usually of embolic origin, resulting in muscle ischemia and pain. Certainly the disease was well advanced when she began to run a fever and complain of pain in the left upper quadrant, approximately five months before entry. As for the course of the disease, I see little worth commenting on at length. The episodes described are quite typical of bacterial endocarditis. Rashes are infrequent in the disease, but they do occur. Possibly it may have been a drug rash due either to penicillin or to something else, but rashes occur without the use of drugs in bacterial endocarditis. I think without much question that the repeated episodes of pain in the left upper quadrant can be ascribed to infarction of the spleen. It is such a common occurrence in the disease that I see no reason for trying to explain it on any other basis. The periumbilical pain, with nausea and anorexia, that persisted for a few days may have been due to an embolus in one of the small mesenteric vessels, too small to produce infarction of the bowel but large enough to produce a temporary disturbance of bowel function. The only findings worth commenting on are those in the left thorax. I think that it would be helpful to look at the x-ray films.

Dr. James R. Lingley: This is a film taken on admission. The heart shadow is not appreciably enlarged in the transverse diameter, but it is enlarged across the base, owing to enlargement of the left auricle. It has a rather typical mitral configuration. The lungs were clear at that time.

Dr. Kranes: Have you any hesitation in saying that it was a rheumatic heart?

Dr. Lingley: That was our interpretation.

Dr. Kranes: That is not quite clear from the record.

Dr. Lingley: I remember it particularly because I interpreted the films myself. This film was taken four days later and one month before death and shows a marked change on the left. There is extensive density in the left lower lung field, with elevation of the diaphragm and displacement of the heart shadow to the left. The appearance is consistent with atelectasis of the left lower lobe, and there apparently is some fluid along the axillary border. The amount of atelectasis is not inconsistent with an infarct.

Dr. Kranes: Can a high diaphragm itself produce that degree of atelectasis?

Dr. Lingley: I do not believe so. I think that the elevation of the diaphragm is due to a collapse of the left lower lobe.

Dr. Kranes: Before seeing the films I had visualized a somewhat different state of affairs. I had the impression, from the abstract, that most of the findings in the chest were explained by fluid, despite the statement that the heart had been displaced to the left. Consequently I had postulated that what was taking place was probably a large infarction of the superior pole of the spleen, as a result of which an inflammatory process had taken place, the spleen becoming adherent to the under surface of the diaphragm, with consequent irritation and outpouring of fluid into the pleural cavity. According to Dr. Lingley, however, this looks more like a process going on within the lung itself. If that is so, one would have to assume that the infarction was pulmonary rather than splenic. I hesitate to make that diagnosis since in doing so one has to assume that the vegetations were on the right side of the heart — a rare occurrence in bacterial endocarditis. The overwhelming majority of vegetations occur either on the mitral or aortic valve, or both. Only infrequently are they found on the tricuspid or pulmonary valve from which pulmonary emboli would originate.

This raises another interesting question. Did this patient have rheumatic heart disease or are the findings described here compatible with a congenital heart of some sort? I think that one has a right to raise that question, despite the history of two attacks of rheumatic fever. The first attack, which occurred at the age of three and during which time the heart murmur was heard, may be seriously questioned. Although rheumatic fever does occur in children below the age of four or five it is extremely rare. When one hears a heart murmur at that age, even in the presence of chorea, one has a right to ask whether the murmur might not have been present previously. There is no way of answering that question in this case. The type of congenital lesion that might explain this would be, of course, a patent interventricular septum, and the findings on admission of a thrill and a loud

systolic murmur are not inconsistent with such a diagnosis. I am, however, inclined to believe the history and to accept at its face value the statement that two attacks of rheumatic fever occurred, particularly since the configuration of the heart, as pointed out by Dr. Lingley, was also consistent with rheumatic heart disease.

The real problem in this case centers around the nature of the terminal episode. What caused this patient's death within a period of thirty minutes after she had been apparently progressing quite satisfactorily? The usual causes of sudden death in patients with bacterial endocarditis are the result of emboli to various vital organs — the brain, the heart or one of the viscera. So far as the brain is concerned, there certainly is no evidence in this terminal picture on which to incriminate the central nervous system, although it is the most frequent site of embolism. They have been extremely explicit in telling us that the patient moved her arms and legs while thrashing around in bed.

So far as the heart is concerned it is somewhat more difficult to exclude a massive myocardial infarct as a result of occlusion of one of the coronary arteries by a vegetation. It is not an infrequent cause of sudden death in cases of bacterial endocarditis and is due either to large vegetations growing over the aortic valve and occluding one of the coronary mouths or to the breaking off of a small vegetation, which lodges inside a coronary artery. Although it is a perfectly possible situation that cannot be excluded, it is rather unlikely because of the clinical findings. The description of this episode does not sound like an attack of acute myocardial infarction. Although it does not specifically state so, I am assuming that the patient had a good deal of pain in the left upper quadrant. The only evidence is that she grasped that area. The record does not state that she had pain, but it is reasonable to infer that there must have been a good deal of pain. Pain localized to the left upper quadrant seems to me unusual in a patient dying of myocardial infarction. If that were the case, one would expect to hear some alteration in the quality of the heart sounds, but they are described as unchanged, aside from an increase in rate. I think that a large myocardial infarct from an occluded coronary vessel is not probable, although I cannot rule it out.

Nor can I exclude a pulmonary embolus. If these previous findings in the chest were due to pulmonary infarcts, the terminal event might also have been due to the same process, but again the clinical picture is not that of a pulmonary embolus. One learns, however, in these exercises that typical clinical pictures are rare. She was described as pale and sweating rather than cyanotic. Furthermore, after a month of therapy with penicillin, if any vegetations did persist, one would not expect them to be large. The pulmonary infarcts that occur with bacterial endocarditis are likely to be small, not the massive ones that cause death. One must, of course, consider the possibility that the embolus arose from thrombi in a leg vein, but there was no evidence of phlebitis except for the pain in the right leg several weeks earlier. I am rather inclined to believe that this patient did not have a pulmonary embolus, although clinically such a diagnosis is somewhat more acceptable than is myocardial infarction.

Another unusual and rare cause of sudden death in bacterial endocarditis is rupture of an aortic valve due to ulceration secondary to the vegetative process. I do not see how we can make that diagnosis here. The usual picture is that of rapidly progressive congestive heart failure, as well as the occurrence of a diastolic murmur that had not previously been present. We are specifically told that no change occurred in the pre-existing murmurs, and under the circumstances a ruptured aortic cusp is extremely improbable.

Could this patient have had an embolus to one of the abdominal viscera? If so, to what viscus? I do not see how an embolus to any of the abdominal viscera usually involved could produce death in thirty minutes. It takes time for infarction of the bowel and perforation with peritonitis to occur. It would be a most unusual state of affairs for a patient with mesenteric thrombosis to die within so short a period. Had it taken place over a period of hours, I should consider it much more seriously.

One would like, of course, to tie up this terminal episode with what had gone before. The patient had complained several times of discomfort in the left upper quadrant, which I assume was due to infarction of the spleen. At the end she suddenly grasped the left upper quadrant, apparently in great pain. I should like to believe that the terminal episode had something to do with the spleen. If so, the only conceivable explanation that I can offer is a spontaneous rupture of the spleen. Certainly the clinical picture as given here fits that much better than anything else I can think of. I hesitate to mention it because it is so rare. It occurs in a variety of septic spleens and in leukemia. Although I am not aware of its having been described in bacterial endocarditis, I know that it has been reported in many other conditions in which the spleen has been septic or infarcted, such as in malaria. Certainly, if this were a case of malaria and the patient suddenly grasped the left upper quadrant and died in thirty minutes with signs and symptoms of shock or hemorrhage, there would be no doubt in one's mind that the patient died of a ruptured spleen. Possibly I have become sensitized to this condition after having seen one case of malaria with a ruptured spleen and a picture not dissimilar to this. It seems to me that the diagnosis of the terminal event lies essentially between pulmonary embolus, which is far more frequent but does not fit the clinical picture too well, and spontaneous

rupture of the spleen, which is rare but fits the description here better than anything else I can think of. So I shall suggest that.

DR. F. DENNETTE ADAMS: Could the lung picture have been due to ordinary collapse of the lung without infarction in a sick patient lying in bed?

DR. KRANES: As the terminal event?

DR. ADAMS: No.

DR. LINGLEY: It would be unusual to have that amount of fluid with collapse.

DR. DONALD S. KING: The symptoms of writhing and groaning interest me. I have never seen that picture with a pulmonary embolus. The patient with a pulmonary embolus is usually quiet and does not writhe. I think Dr. Kranes's suggestion that it was due to something else is a good one.

DR. BENJAMIN CASTLEMAN: Dr. FitzHugh, I believe that you saw this patient. Will you say a word or two about your impression?

DR. GREENE FITZHUGH: First, I want to congratulate Dr. Kranes on his excellent discussion. We thought that the symptoms in the left upper quadrant were due to infarction of the spleen or left kidney. The spleen was felt by only one person out of the six or eight who examined her. I was not that one. We believed that she was having repeated infarcts. The lungs were negative.

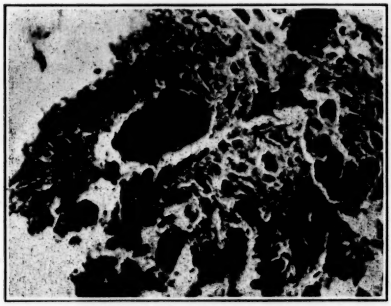


FIGURE 2. *Photomicrograph of a Healing Bacterial Vegetation on the Mitral Valve.*
The dark-staining masses are clumps of cocci. Note the giant cell.

CLINICAL DIAGNOSES

Subacute bacterial endocarditis.
Pulmonary embolus.

DR. KRANES'S DIAGNOSES

Subacute bacterial endocarditis.
Splenic infarcts.
Spontaneous rupture of spleen.

ANATOMICAL DIAGNOSES

Subacute bacterial endocarditis: mitral valve.
Splenic infarcts, with rupture of spleen.
Hemoperitoneum.
Rheumatic heart disease, with mitral and aortic involvement.
Embolic nephritis, healing.
Hydrothorax, left.
Pulmonary atelectasis: left lower lobe.
Gluteal abscesses.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: At autopsy, when the abdominal cavity was opened, we found the entire left side filled with clotted blood. The blood was present in both the lateral gutter and the pelvis, but it was particularly concentrated in the region of the hilus of the spleen. There were infarcts in both the upper and the lower poles of the spleen. Between them, just anterior to the hilus, was a tear with a reddish-black clot over it. The spleen was about three to four times normal in size, weighing 640 gm. Most of the enlargement was probably due to the large amount of intrasplenic hemorrhage, which formed a clot measuring about 10 cm. in diameter. This hemorrhage was adjacent to both the upper and lower infarcts; we were not certain that the hemorrhage began in either of these infarcts, although it probably did. There was no evidence of infarction in the central portion of the spleen.

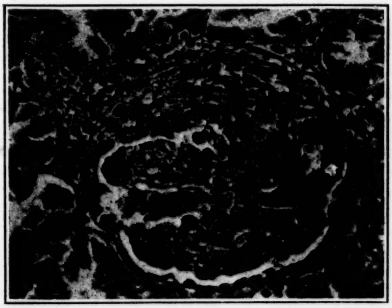


FIGURE 3. *Photomicrograph Showing a Healing Process in the Upper Portion of a Glomerulus.*

The heart was only slightly hypertrophied. There was slight thickening of the aortic valve, but no evidence of bacterial endocarditis. The mitral valve was markedly thickened, and on one of the leaflets was a hard vegetation 2 or 3 mm. in diameter, superimposed on which was a thin layer of friable

tissue. Clumps of organisms could be seen microscopically within the vegetation, with a lot of scarring and granulation tissue around them. In one granulating area we found a giant cell of the foreign-

was fairly adherent to the diaphragm. Dr. Kranes's initial hypothesis was therefore correct. The infarct had broken down and had irritated the diaphragm and pleura, thus producing pleural effusion

FIGURE 4. *Photomicrograph Showing the Necrosis and Foreign-Body Giant-Cell Reaction at the Site of the Injection of Penicillin in Beeswax and Peanut Oil.*

body type (Fig. 2). The process, therefore, was almost healed but still contained many organisms.

The kidney showed evidence of a healing embolic nephritis. About one fifth or one sixth of the glomeruli were involved; in some there was complete involvement, but in most of them only a small portion of the tuft was infarcted (Fig. 3). There was no evidence of an acute embolic nephritis: all the lesions were healing or healed, which is fairly good evidence that no further emboli were getting into the kidney. This patient had been receiving penicillin in beeswax and peanut oil, and a section of the buttocks, where the injections had been given, showed fairly extensive muscle degeneration, surrounded by a foreign-body reaction (Fig. 4). I should think that this might have caused a lot of pain.

DR. KRANES: Was there any evidence of pulmonary infarcts?

DR. CASTLEMAN: In the left lower chest there was 200 cc. of clear, straw-colored fluid, with surrounding atelectasis of the left lower lobe. There was no evidence of infarction. The splenic infarct in the upper pole was cystic, and before sectioning felt as if it might have been an abscess. This had extended to the capsule of the spleen and

and atelectasis. The pleural effusion had disappeared for the most part by the time of autopsy. This last x-ray (Fig. 1) was taken one month before death.

CASE 32192

PRESENTATION OF CASE

First admission. A fifty-eight-year-old housewife entered the hospital during an acute asthmatic attack.

About twenty-five years before admission the patient had her first attack of bronchial asthma. There was no known precipitating event. Subsequently she had mild intermittent attacks for three or four years. Then she inhaled vapors from boiling hemlock branches and had a complete remission for fifteen years. Four years before admission, at the age of fifty-four, following an emotional upset she was again stricken by an asthmatic attack. After that there were infrequent mild attacks until ten months before admission, when she had an attack severe enough to require treatment by a doctor. Her next severe attack brought her to the hospital.

On physical examination there was labored, wheezing respiration, with a prolonged expiratory phase. The anteroposterior diameter of the chest was increased. The lung fields were hyper-resonant, the diaphragm was depressed, and expansion diminished.

The temperature was 99°F., the pulse 110, and the respirations 22. The blood pressure was 160 systolic, 110 diastolic.

The red-cell count was 5,360,000, and there were 15 gm. of hemoglobin. The white-cell count was 10,950, with 62 per cent neutrophils and 9 per cent eosinophils. The urine was normal. By x-ray examination the lung fields were bright. The diaphragm was low, and its excursion limited. The pulmonary markings were prominent throughout the chest, particularly at the bases. The heart was slightly enlarged to the left. Orris powder caused a 2-cm. skin wheal, with a 1-cm. erythematous border. Chicken feathers, ragweed and goose feathers also gave positive, although smaller, skin reactions.

The asthmatic attack responded to aminophylline, ephedrine and sedatives. The patient was discharged on the third hospital day.

Final admission (twenty-eight months later). Within the next two years there were seven brief admissions for asthma. All were completely or partially relieved by aminophylline, chloral hydrate and potassium iodide. Between admissions the patient was sometimes free of asthma, but she often had attacks that required adrenalin injections by a doctor or visiting nurse. There was a general trend toward chronic asthma, with gradual shortening of the periods of good health. After several of the admissions she spent one or two weeks in a nursing home. During her third admission she had a pneumonia affecting the right lower lobe, and a Type 3 pneumococcus was isolated from the sputum. At the end of the two-year period she carried on fairly comfortably by taking potassium iodide, aminophylline and ephedrine daily. There was never any evidence of heart failure, and an electrocardiogram on the next to the last admission was normal. The chest films showed little change in the two years.

Two weeks before the final admission she began to have more frequent attacks, particularly at night. They gradually became worse, until finally nothing relieved them.

Physical examination revealed slight cyanosis and moderate respiratory distress. Expiratory wheezes and rhonchi were heard over both lung fields but were more numerous on the left. The heart sounds were faint. The pulmonic second sound was greater than the aortic. The neck veins were not distended. There was no enlargement or tenderness of the liver, nor any ankle edema.

Twenty-four hours after admission the patient's condition was considerably improved under the usual treatment of 10 cc. of aminophylline intravenously. While dressing to go home, she suddenly became much worse. When examined a few minutes later, she was seated half dressed in a chair, doubled forward and breathing in short grunts. The usual asthmatic rales were heard at the right base posteriorly, but the respiratory exchange was very shallow. She was extremely cyanotic. Oxygen was given by mask, and artificial respiration was started as the breathing was reduced to an occasional gasp. There were several convulsive movements of the face, and she died ten minutes after the onset of the attack.

Differential Diagnosis

Dr. J. Evarts Greene: Here is another case in which the cause of sudden death is the chief problem. We know that this woman had asthma and that it began twenty-five years before admission, at the age of thirty-three years. She had a little trouble for three or four years, and then the inhalation of hemlock vapor "cured" her. At the age of fifty-four, following an emotional upset, she again developed asthma. This brings up the possibility of a reflex or nervous factor in the precipitation of asthmatic attacks, and apparently this patient did have asthma in response to emotional stimuli.

Physical examination on the first admission gave good evidence of emphysema. The anteroposterior diameter of the chest was increased, the lung fields were hyper-resonant, the diaphragm was depressed, and its expansion was diminished. At that time the blood pressure was 160 systolic, 110 diastolic, which indicates that she probably had some hypertension.

The laboratory work did not show a marked increase of red cells or hemoglobin, so that it does not appear that she had any compensatory polycythemia. The x-ray film of the chest was said to show prominent pulmonary markings, particularly at the bases. The heart was slightly enlarged to the left, and there was a question whether she had pulmonary fibrosis. The patient did obtain relief by coming to the hospital on frequent occasions. It is possible that she was sensitive to some dust at home, which was avoided when she came to the hospital.

She had Type 3 pneumococcus pneumonia on one of her admissions. In our experience pneumococcal pneumonia is infrequent in patients with bronchial asthma. Of course, it can and does happen.

Two weeks before the final admission, the attacks increased in frequency and severity. On entry she was slightly cyanotic and musical rales and rhonchi were heard, more numerous on the left side than on the right. There was no evidence of myocardial failure: the neck veins were not distended and there was no enlargement of the liver or edema of the ankles. Apparently no moist rales were heard in the lungs. Presumably no x-ray film of the chest was taken at the onset of the terminal event, which only lasted ten minutes. Perhaps we should see the x-ray films.

DR. JAMES R. LINGLEY: We have films over a two-year period, which show practically the same thing, except for the pneumonia. This is the first film, and it shows marked emphysema on both sides, with slight enlargement of the heart on the left. This is the last film, taken two years later, and it shows practically no change.

DR. GREENE: Is there any appreciable sign of fibrosis?

DR. LINGLEY: There may be a moderate degree.

DR. GREENE: Then we come to the terminal event, which lasted only ten or fifteen minutes. The cause of sudden death is difficult to determine. I do not feel entirely satisfied with any of the various possibilities that I think should be considered. The first is that this patient died simply of a bad attack of asthma, which came on for no obvious reason just as she was getting dressed to go home. Perhaps it was in response to the exertion of getting dressed, or perhaps there was an emotional basis in the thought of going home. Asthmatic attacks do develop suddenly under these conditions. On the other hand, a ten-minute period is extremely short for death from an attack of bronchial asthma, and apparently she had been fairly comfortable just before the terminal event. Cases have been described in which asthma led to death within an extremely short period of time.[1] I believe it is possible that this patient's death was based purely on asthma, although it is unlikely.

The second possibility that comes to mind is a massive pulmonary embolus. I cannot recall having seen fatal pulmonary embolism in patients with asthma. We have no evidence from the history that there was any focus from which a pulmonary embolus might have come, but there is no reason why she may not have had a large embolus from a femoral or pelvic vein.

A third possibility, coronary thrombosis, seems quite unlikely. There is no mention that the patient had pain. The main complaints seem to have been dyspnea and cyanosis, so that although coronary thrombosis is possible, I believe that it is unlikely.

A fourth possibility is spontaneous pneumothorax. A few cases have been described in which asthmatic patients have had spontaneous pneumothorax followed by death,[2] but they are exceedingly rare. If such had occurred one would expect the patient to have had a good deal of pain, and there should have been typical signs of pneumothorax, with shifting of the heart and the mediastinum to the opposite side. On the other hand, the description of the examination of the chest during the terminal episode is not at all complete, and we cannot be quite sure about the shift of the mediastinum, or whether the intercostal spaces were larger on the one side than on the other.

The fifth and final possibility is massive collapse of the lung, which may be due to sudden obstruction of the bronchi, to pulmonary infarction, to infection, of which we have no evidence, to an allergic cause or to nervous stimuli. Again there should have been, unless the massive collapse was bilateral, shift of the mediastinum, although this time toward the affected side, and a high diaphragm on the same side.

Finally, I might mention just for the sake of completeness the fact that some patients with asthma die following the administration of morphine. We have no indication that this patient had been given morphine. Death has also been due to periarteritis nodosa and asthma, but there is no evidence that this patient had periarteritis nodosa.

Granted that none of the explanations that I have offered for the terminal episode are entirely satisfactory, I favor a diagnosis of pulmonary embolism in this case, particularly because of the sudden onset and the rapid course of the terminus, but also because the patient was making some exertion in the act of getting dressed, perhaps the first she had made in some hours, which could have led to the dislodging of a large thrombus in a femoral or pelvic vein.

CLINICAL DIAGNOSIS

Bronchial asthma.
Pulmonary embolus?
Pulmonary emphysema.

DR. GREENE'S DIAGNOSES

Bronchial asthma.
Pulmonary emphysema.
Hypertension.
Massive pulmonary embolism.

ANATOMICAL DIAGNOSES

Bronchial asthma.
Spontaneous pneumothorax, left.
Pulmonary emphysema.
Cor pulmonale.

PATHOLOGICAL DISCUSSION

DR. BENJAMIN CASTLEMAN: The autopsy findings in the usual case of a patient dying of an asthmatic paroxysm have often been described at these exercises by Dr. Mallory, and we have had 18 such cases out of a series of autopsies on 50 asthmatic patients. The lungs are usually ballooned and meet in the midline, covering the heart. They do not collapse when the chest plate is removed. On section, the bronchi are filled with mucous plugs, usually in the medium-sized bronchi, although occasionally in the large bronchi. The walls of the bronchi are thickened, and the mucous glands are hypertrophied. The basement membrane is thickened, and there is lymphocytic and eosinophilic cellular infiltration of the walls. Autopsy of this patient

showed all these findings in the lungs, but in addition, when the chest plate was removed, a large puff of air came out of the left chest and the left lung was found way over to the right against the spine. She undoubtedly had, as Dr. Greene mentioned, a pneumothorax. We have here then a combination of two conditions—the type of lung that is found in an asthmatic paroxysm and a pneumothorax. I believe that the latter was probably the precipitating cause of death. This is the first case of pneumothorax as a complication of an asthmatic paroxysm that we have had.

DR. RACKEMANN: I should like to ask Dr. Lingley if he would look at the x-ray films again to see if he can find an emphysematous bleb. Why did the lung rupture?

DR. LINGLEY: There is no visible evidence of a bleb in this film.

DR. CASTLEMAN: The left lung showed two or three emphysematous blebs, but we were unable to find a definite tear. There was a moderate degree of emphysema throughout the lung, and definite hypertrophy of the right ventricle.

REFERENCES

1. Rackemann, F. M. *Clinical Allergy Particularly Asthma and Hay Fever: Mechanism and treatment.* 617 pp. New York: Macmillan Company, 1931. P. 421.
2. Faulkner, W. B., Jr., and Wagner, R. J. Fatal spontaneous pneumothorax and subcutaneous emphysema in asthmatic: report of case with bronchoscopic findings. *J. Allergy* 8:267-272, 1937.

The New England
Journal of Medicine

Formerly
The Boston Medical and Surgical Journal
Established 1828

Owned by the Massachusetts Medical Society and Published under the Jurisdiction of the Committee on Publications

Richard M. Smith, M.D., *Chairman*
James P. O'Hare, M.D. Conrad Wesselhoeft, M.D.
Oliver Cope, M.D. John Fallon, M.D.

Official Organ of
The Massachusetts Medical Society
and
The New Hampshire Medical Society

Editorial Board
Joseph Garland, M.D. A. Warren Stearns, M.D.
Shields Warren, M.D. Chester S. Keefer, M.D.
C. Guy Lane, M.D. Fletcher H. Colby, M.D.
William A. Rogers, M.D. Robert L. Goodale, M.D.
Henry R. Viets, M.D. Chester M. Jones, M.D.
Robert M. Green, M.D. Harvey R. Morrison, M.D.
Charles C. Lund, M.D. Maxwell Finland, M.D.

Associate Editors
Thomas H. Lanman, M.D. Donald Munro, M.D.
Henry Jackson, Jr., M.D.
Walter P. Bowers, M.D., Editor Emeritus
Robert N. Nye, M.D., Managing Editor
Clara D. Davies, Assistant Editor

Subscription Terms: $6.00 per year in advance, postage paid, for the United States (medical students, $3.50 per year); Canada, $7.04 per year (Boston funds); $8.52 per year for all foreign countries belonging to the Postal Union.

Material should be received not later than noon on Thursday, two weeks before date of publication.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston 15, Massachusetts.

COMING ANNUAL MEETING

Following an enforced hiatus of two years, the one hundred and sixty-fifth anniversary of the Massachusetts Medical Society will be held at the Hotel Statler, Boston, on May 21, 22 and 23; the program of the meeting appears elsewhere in this issue of the *Journal*.

The first general session will begin at 2:00 p.m. on May 21 and comprises seven papers of general interest. Late that afternoon there will be a meeting of the supervising censors, followed in the evening by the annual meeting of the Council.

On the following morning, following four papers on miscellaneous topics, the annual meeting of the Massachusetts Medical Society will be held, the annual oration to be given by Dr. Frank H. Lahey. The afternoon session begins with five papers of general interest and ends with the Shattuck Lecture, which will be given by Colonel John B. Youmans, M.C., A.U.S., chief, Nutrition Branch, Office of the Surgeon General, and professor of medicine, Vanderbilt University School of Medicine, the title being "Nutrition and the War." The annual dinner of the Society will be held that evening; the president, Dr. Reginald Fitz, will preside, the guest speakers being Dr. Roger I. Lee, president of the American Medical Association, and Dr. Elmer S. Bagnall, past president of the Society.

The program for the morning of the third day lists three papers of general interest and a symposium on bleeding from the gastrointestinal tract. At noon, the various sections will hold luncheon meetings, at each of which a short talk will be given on an appropriate topic. The meeting will conclude with the afternoon session, the program consisting of seven papers on miscellaneous topics.

The scientific and technical exhibits will be larger than ever before. Tremendous advances in the diagnosis and treatment of disease were made during the war years, and many of these are now being made available to the general practitioner for the first time. For this reason all the exhibits, particularly the technical ones, deserve the close attention of those who attend the meeting.

The usual continuous motion-picture program is scheduled for the second and third days.

An interesting schedule has been arranged for the wives of members, including attendance at the Pop Concert at Symphony Hall on the evening of the first day. A golf tournament for members will be held at the Wollaston Golf Club on the afternoon of the second day.

The Committee on Arrangements, which is composed of Dr. Roy J. Heffernan, chairman, and Drs. G. Guy Bailey, Jr., Harold G. Giddings, Robert L. Goodale and Sidney C. Wiggin, has set forth a program that should appeal to the returned veteran and to the general practitioner, and it seems likely that all previous records of attendance will be shattered.

THE INADEQUACIES OF MEDICAL CARE

V. How They Can Be Corrected

This final editorial summarizes the suggestions made in the previous editorials concerning the correction of existing inadequacies of medical care. These comments will not be listed according to particular aspects but according to the agency that is responsible for their accomplishment.

The Government should promote an economy that guarantees good wages and a high standard of living, including adequate housing and transportation facilities. It should establish grants-in-aid to states in which there is demonstrated need for facilities concerned with the health of the people; incidentally, this could best be accomplished by the creation of a national department of health, headed by a secretary of Cabinet rank. Through an extension of the Social Security Act it should provide compensation for loss of wages due to sickness. Finally, it should organize, supervise and support a scheme for the continuance of co-ordinated medical research.

State governments should take the necessary steps, statutory or otherwise, to assure the licensing of well trained physicians, to eliminate improper medical practice by cultists, to improve hospital standards, to guarantee the necessary funds for the care of the indigent and to encourage an increase in nursing personnel through the licensing of attendant nurses. They should construct and equip hospitals and health centers in areas of demonstrated need and, if necessary, should employ or subsidize physicians in these institutions, all with the assistance of federal grants-in-aid. They should educate the public concerning matters of diet, accepted methods of preventive medicine and available facilities for medical care.

Medical schools should pay more attention to the need for training physicians who qualify as general practitioners rather than as specialists.

Both national and state medical societies should sponsor comprehensive programs for postgraduate medical education. State medical societies should assist state departments of health in educating the public regarding preventive medicine and available medical facilities, chiefly through the medium of district and local health councils, and should promote plans for the assistance of those physicians who have had a substandard education.

Physicians should keep themselves professionally "fit" and should limit their practice to cases for which they are qualified to care. In certain instances, they should consider the advantages offered by group practice.

The number of hospital beds must be increased, and as a temporary expedient, hospitals should make full use of existing facilities. If necessary, they should improve equipment and personnel to meet accepted minimum standards. They should seriously consider the advisability of establishing affiliated convalescent homes.

All these things would do much to improve the health of the people, *but* they would not appreciably lower the cost of medical care. In other words, people must still insure against the expense of catastrophic illness, and the mere fact that they do has little to do with assuring good medical care. Indeed, a healthier Nation can only result from the combined efforts of a variety of agencies — governmental and otherwise. For this reason there seems to be little excuse for maintaining that a nation-wide system of compulsory health insurance is the *only* solution to the problem. It is true that such a scheme would solve one of the difficulties, — equalization of the costs of medical care, — but at the same time it would seriously handicap, if not abolish, many existing enterprises that are gradually accomplishing the very things that contribute to the improvement of medical care. Such enterprises, particularly voluntary schemes for prepayment medical-care insurance, should be encouraged and promoted by the medical profession and by the public, and they should be supported by states, cities and towns, if necessary through federal grants-in-aid.

MASSACHUSETTS MEDICAL SOCIETY

DEATH

FESSENDEN — Charles H. Fessenden, M.D., of Newton, died April 19. He was in his eighty-third year.

Dr. Fessenden received his degree from Boston University School of Medicine in 1886. Until his retirement ten years ago, he had been for many years an x-ray specialist at Newton Hospital.

His son, two grandchildren and two great grandchildren survive.

MEDICOLEGAL ABSTRACT

Regulation of Practice by Government: Unauthorized practice. The defendant was a licensed cosmetologist and operated a "clear-skin" institute. He was charged with practicing medicine without a license in violation of Michigan Comp. Laws 1929, Paragraphs 6743 and 6745. A jury found him guilty, and he appealed. The judgment was affirmed. The evidence showed that the complaining witness was suffering from acne and that the defendant, for a fee, undertook to treat her and did treat her by means of applying ointment and extracting blackheads. A physician qualified as an expert on skin diseases testified that papular acne, "ordinarily spoken of as pimples," was a disease a physician without special training in dermatology would find difficult to differentiate from secondary syphilis and that it would certainly require a physician to treat it. There was some other evidence that the defendant engaged in the practice of medicine in violation of the statute. — *People* v. *Gilbert*, 312 Mich. 320, Oct. 8, 1945.

This case, however, is particularly interesting because it calls attention to the exceedingly broad provisions of the Michigan statute under which the conviction was obtained. Paragraph 6743 of the Comp. Laws of 1929 makes it a misdemeanor to practice medicine without a license. Paragraph 6745 defines the practice of medicine as the actual diagnosing, curing or relieving in any degree, or professing or attempting to diagnose, treat, cure or relieve any human disease, ailment, defect or complaint, whether of physical or mental origin, by attendance or advice or by prescribing or furnishing any drug, medicine, appliance, manipulation or method, or by any therapeutic agent whatsoever. One has no need to be a lawyer to see how sweeping would be a literal construction of the provision making it criminal "to relieve by advice any human complaint of mental origin."

Although a chiropractor is not under Michigan law a physician or surgeon (*Erdman* v. *Great Northern Life Ins. Co.*, 253 Mich. 579), he is licensed under other provisions of the statute so that he can practice medicine. — *Locke* v. *Ionia Circuit Judge*, 184 Mich. 535 and *People* v. *Lewis*, 233 Mich. 240. Similarly pharmacists are separately licensed. Although nurses are not specially licensed in Michigan they would seem to be within the principle that treatment given under supervision and direction of a doctor is not in violation of the statute. — *In re Carpenter's Estate*, 196 Mich. 561. The practice of midwifery alone, not accompanied by any other practice of medicine and surgery, is also excluded by interpretation.

Other limitations are not so clear. In *Locke* v. *Ionia Circuit Judge*, 184 Mich. 525, at 539, the court said:

> This sweeping effort at definition, with all provisions "otherwise" taken into account would render criminal numerous gratuitous and humane acts of relief and kindness to the suffering common amongst mankind in all ages and places. The police power of the state, though comprehensive, is scarcely adequate to compass the possibilities of such a definition, and it is difficult to discern in the title of the act any warning of a purpose to make such definition a part of the law of this commonwealth.

In *People* v. *Watson*, 196 Mich. 34, the court said that some acts covered by the language of the statute might be innocently done. The assumption that things so done would not be in violation of the statute is clear. The meaning of things "innocently done" is not so clear. Certainly no criminal intent or *mens rea* is required to constitute the crime penalized by the statute. The court did, however, refer with approbation to the exception of "gratuitous and humane acts" made above. The notion that the statute would be limited to those who engage in the practice of medicine for gain and as a vocation is further strengthened in *People* v. *Eckelyn*, 217 Mich. 341. The nature of the acts done, however, would seem to be much more relevant than the question of whether they were done for charity or for pay. Thus, performing an appendectomy is practicing medicine even when done for charity, while consoling a widow could hardly be so considered even if were done in pursuit of a vocation.

Despite the broad language of the act there are extremely few cases under it, limitation in its meaning apparently depending primarily on the good sense of the prosecuting authorities.

MISCELLANY

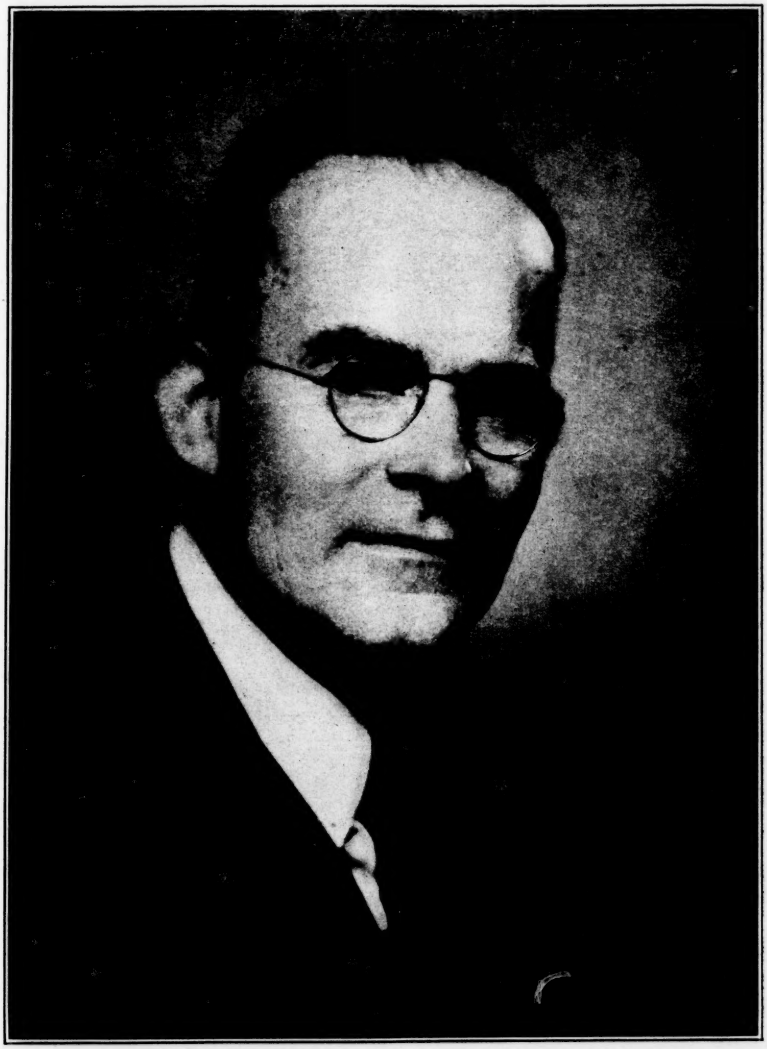
PASSANO FOUNDATION AWARD

Selection of Dr. Ernest W. Goodpasture, professor of pathology and dean of the School of Medicine of Vanderbilt University, Nashville, Tennessee, as the 1946 recipient of the Passano Foundation Award is announced by the Board of Directors of the Foundation. Presentation of the $5000 cash award will be made at an appropriate ceremony in Osler Hall of the Medical and Chirurgical Faculty of Maryland, in Baltimore, on the night of May 15. The Foundation, which was established in 1944 by the Williams and Wilkins Company, of Baltimore, proposes to aid in any way possible the advancement of medical research, especially research that bears promise of clinical application. For the encouragement of such research the Foundation has established the award as one of its activities. Following the presentation of the award by Mr. Edward B. Passano, chairman of the Board of Directors of the Williams and Wilkins Company, Dr. Goodpasture will deliver an address entitled, "Research and Medical Practice." Dr. Goodpasture receives the award for his original development of the method for propagation of viruses in pure culture by inoculation of chick embryos and for his outstanding contributions to advancement of knowledge of the cell-parasite relation in bacterial and viral infections.

DR. O'HARA APPOINTED DEAN AT TUFTS

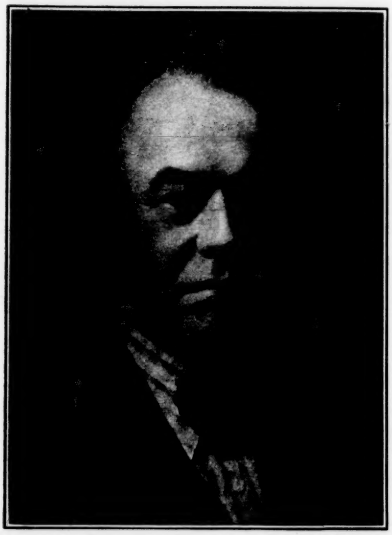
Dr. Dwight O'Hara, of Waltham, who is president-elect of the Massachusetts Medical Society, has recently been appointed dean of Tufts College Medical School. Dr. O'Hara has been professor of preventive medicine at the school for a number of years and has been acting dean for the past four years, in the absence of Dr. A. Warren Stearns, who served as a captain in the Medical Corps of the United States Navy. Dr. Stearns has been made head of the newly created Department of Applied Sociology at the Tufts School of Liberal Arts.

OFFICERS OF THE MASSACHUSETTS MEDICAL SOCIETY, 1945—1946



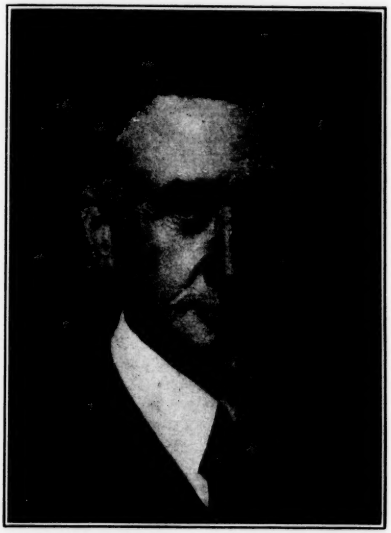
Bachrach

DR. DWIGHT O'HARA, *President-Elect*



Waid Studio

DR. REGINALD FITZ, *President*

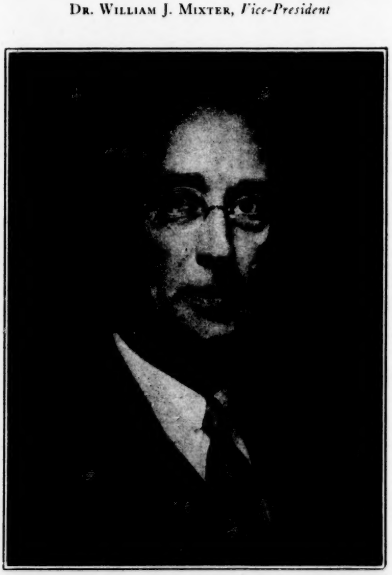


Kaiden-Keystone

DR. WILLIAM J. MIXTER, *Vice-President*

Taylor Sargent

DR. MICHAEL A. TICHE, *Secretary*



Backrach

DR. ELIOT HUBBARD, JR., *Treasurer*

PROGRAM OF THE ONE HUNDRED AND SIXTY-FIFTH ANNIVERSARY OF THE MASSACHUSETTS MEDICAL SOCIETY

Tuesday, Wednesday and Thursday, May 21, 22 and 23, Hotel Statler, Boston

The Registration Desk will be located on the Mezzanine Floor, and all who attend the meeting are requested to register.

Owing to the lack of personnel and food supplies, the Hotel Statler must be informed concerning the number of members who will attend the various luncheons and dinners. Attendance is not limited, but tickets *must* be obtained in advance.

Members of the Society who are not councilors may obtain tickets for the Pop Concert on Tuesday evening by purchasing them ($1.50 each) from Society Headquarters, 8 Fenway, Boston 15, *on or before* May 15. Their wives and friends are cordially invited to attend.

TUESDAY AFTERNOON, MAY 21

First General Session

GEORGIAN ROOM

DR. RALPH R. STRATTON, *Chairman*
DR. CARL BEARSE, *Co-chairman*

2:00 *Foreign Bodies in the Respiratory Tract.* DR. JOHN R. RICHARDSON, Boston. Assistant surgeon, Massachusetts General Hospital and Massachusetts Eye and Ear Infirmary; bronchoscopist, Massachusetts Eye and Ear Infirmary.

2:25 *The Problem of Venous Thrombosis and Pulmonary Embolism.* DR. JOHN HOMANS, Boston. Clinical professor of surgery (emeritus), Harvard Medical School.

2:50 *Anal Pruritus.* DR. NEIL W. SWINTON, Waban. Surgeon, Lahey Clinic and New England Deaconess and New England Baptist hospitals.

3:15 *Anoxia Neonatorum.* DR. CHARLES L. SULLIVAN, Chestnut Hill. Assistant obstetrician, Massachusetts General and Saint Elizabeth's hospitals.

3:40 *Progress — Penicillin Inhalation Therapy.* DR. MAURICE S. SEGAL, Boston. Assistant professor of medicine, Tufts College Medical School; junior visiting physician, Boston City and Beth Israel hospitals.

4:05 *Functional Gastrointestinal Disorders: Lessons learned from military medicine.* DR. JAMES A. HALSTED, Dedham. Assistant physician, Massachusetts General Hospital.

4:30 *Tuberculosis Case Findings.* DR. JOHN A. FOLEY, Boston. Director 5th and 6th Medical Services, Boston City Hospital; chief of staff, Boston Sanatorium; clinical professor of medicine, Boston University School of Medicine. DR. JOHN B. ANDOSCA, Boston. Instructor in medicine, Boston University School of Medicine.

5:00 **Supervising Censors' Meeting** (PARLOR D).

6:00 **Cotting Supper for Councilors** (PARLORS A AND B).

TUESDAY EVENING, MAY 21

7:00 **Annual Meeting of the Council** (GEORGIAN ROOM).

8:30 **Pop Concert** (SYMPHONY HALL).

WEDNESDAY MORNING, MAY 22

Second General Session

GEORGIAN ROOM

DR. W. JASON MIXTER, *Chairman*
DR. CHARLES J. E. KICKHAM, *Co-chairman*

9:00 *Some Observations on the Recent Food Rationing Program.* DR. JOSEPH GARLAND, Boston. Physician to the Children's Medical Department, Massachusetts General Hospital.

9:25 *The Diagnosis and Treatment of Infective Hepatitis.* DR. WILLIAM C. MOLONEY, Boston. Assistant professor of clinical medicine, Tufts College Medical School; visiting physician, Carney and St. Elizabeth's hospitals.

9:50 *Tropical Diseases in World War II Returnees.* CAPTAIN JAMES J. SAPERO (MC), U.S.N., Washington, D. C.

10:15 *Streptomycin.* DR. CHESTER S. KEEFER, Boston. Director, Evans Memorial, Massachusetts Memorial Hospitals; physician-in-chief, Massachusetts Memorial Hospitals.

11:00 **Annual Meeting of the Massachusetts Medical Society** (GEORGIAN ROOM).

Annual Oration (following annual meeting): Gastric Surgery. DR. FRANK H. LAHEY, Boston. Director, Lahey Clinic; surgeon-in-chief, New England Deaconess and New England Baptist hospitals.

Annual Luncheon (PARLORS A, B AND C): tickets must be procured in advance of the meeting.

WEDNESDAY AFTERNOON, MAY 22

Third General Session

GEORGIAN ROOM

DR. ELMER S. BAGNALL, *Chairman*
DR. JOHN FALLON, *Co-chairman*

2:00 *Medical Aspects of Hypertension.* DR. NORMAN A. WELCH, Boston. Clinical professor of medicine, Tufts College Medical School; physician-in-chief, Carney Hospital.

2:25 *Results of Bilateral Supradiaphragmatic Splanchnicectomy for Arterial Hypertension.* Dr. Max A. Peet, Ann Arbor, Michigan. Professor of surgery, University of Michigan; chief, Neurosurgical Unit, University Hospital.

2:50 *Delayed Chemical Pneumonitis.* Dr. Harriet L. Hardy, Boston. Physician to Division of Occupational Hygiene, Department of Labor and Industries, Commonwealth of Massachusetts; assistant in medicine, Massachusetts General Hospital.

3:15 *Diabetes in Massachusetts.* Dr. Elliott P. Joslin, Boston. Medical director, George F. Baker Clinic, New England Deaconess Hospital.

3:40 *Surgical Problems in the Management of a Diabetic Patient.* Dr. Leland S. McKittrick, Boston. Surgeon-in-chief, Palmer Memorial Hospital; visiting surgeon, Massachusetts General Hospital.

4:00 **The Shattuck Lecture.** Nutrition and the War. Colonel John B. Youmans, M.C., A.U.S. Chief, Nutrition Branch, Office of the Surgeon General, Washington, D. C.; professor of medicine, Vanderbilt University School of Medicine.

1:00 – 5:00 **Annual Golf Tournament** (Wollaston Golf Club, Quincy, Massachusetts) — Dr. Joseph H. Carey, Chairman.

WEDNESDAY EVENING, MAY 22

7:00 **Annual Dinner of the Massachusetts Medical Society** (Georgian Room).

Presiding:

Dr. Reginald Fitz, president, Massachusetts Medical Society.

Guest Speakers:

Dr. Roger I. Lee, president, American Medical Association.

Dr. Elmer S. Bagnall, past-president, Massachusetts Medical Society.

Ladies are cordially invited to attend the dinner.

Tickets for the dinner *must* be procured in advance of the meeting.

THURSDAY MORNING, MAY 23

Fourth General Session

Georgian Room

Dr. Archibald McK. Fraser, *Chairman*
Dr. Arthur J. Gorman, *Co-chairman*

9:00 *The Hospital as a Center of Education.* Dr. Walter G. Phippen, Salem. Chief of the Surgical Service, Salem Hospital.

Discussers: Dr. H. Quimby Gallupe, Waltham, and Dr. Charles F. Wilinsky, Boston.

9:25 *The Initial Care of the Injured.* Dr. Gordon M. Morrison, Boston. Chairman, Massachusetts Fracture Committee, American College of Surgeons.

Symposium: Bleeding from the Gastrointestinal Tract

9:50 *Gastrointestinal Bleeding: Diagnostic and therapeutic considerations.* Dr. Chester M. Jones, Boston. Physician, Massachusetts General Hospital; clinical professor of medicine, Harvard Medical School.

10:15 *Surgical Aspects of Hemorrhage in Peptic Ulcer.* Dr. George Heuer, New York City. Professor of surgery, Cornell University Medical College; surgeon-in-chief, New York Hospital.

10:40 *The Position of the Roentgenologist in Handling Patients Bleeding from the Gastrointestinal Tract.* Dr. Richard Schatzki, Boston. Radiologist, Massachusetts General Hospital.

11:15 *Question-and-Answer Period.*

11:30 *Comparative Uses of Pentothal Sodium in Military and Civilian Practice.* Dr. Ralph M. Tovell, Hartford, Connecticut. Director of anesthesia, Hartford Hospital. Dr. Charles Barbour, member of staff, Hartford Hospital.

THURSDAY NOON, MAY 23

Section Meetings and Luncheons

12:00 M. — 2:00 P.M.

Tickets for all luncheons *must* be purchased in advance.

Section of Medicine

Parlor B, Hotel Statler

Dr. Albert A. Hornor, Boston, *Chairman*
Dr. Daniel J. Ellison, Lowell, *Vice-chairman*
Dr. Francis C. Hall, Boston, *Secretary*

An Informal Talk on Heart Disease, Stressing Facts We All Want to Remember. Dr. William D. Stroud, Philadelphia. Professor of cardiology, University of Pennsylvania Graduate School of Medicine; associate in medicine, University of Pennsylvania School of Medicine; cardiologist to the Pennsylvania Hospital.

Section of Surgery

Salle Moderne, Hotel Statler

Dr. Charles F. Twomey, East Lynn, *Chairman*
Dr. Alexander J. A. Campbell, Boston, *Secretary*

The Preoperative and Postoperative Care of the Surgical Patient. Dr. Owen H. Wangensteen, Minneapolis. Chief, Department of Surgery, University of Minnesota Medical School.

Section of Pediatrics

The Junior League, Zero Marlborough Street

Dr. Floyd R. Smith, Pittsfield, *Chairman*
Dr. Gerald N. Hoeffel, Cambridge, *Secretary*

The Use and Abuse of Endocrine Therapy in Children. Dr. Nathan B. Talbot, Brookline. Assistant physician, Children's Medical Service, Massachusetts General Hospital.

Section of Obstetrics and Gynecology

Parlor A, Hotel Statler

Dr. Arthur F. G. Edgelow, Springfield, *Chairman*
Dr. William J. McDonald, Boston, *Vice-chairman*
Dr. George Van S. Smith, Brookline, *Secretary*

The Pathologist Looks at Abortion. Dr. Arthur T. Hertig, Boston. Assistant professor of obstetrics and assistant professor of pathology, Harvard Medical School; pathologist, Free Hospital for Women and Boston Lying-in Hospital.

Section of Dermatology and Syphilology

Hancock Room, Hotel Statler

Dr. Bernard Appel, Lynn, *Chairman*
Dr. Fenner A. Chace, Fall River, *Secretary*

Fatty Acid Treatment of Fungous Infections. Dr. Samuel M. Peck, New York City. Associate attending dermatologist, Mount Sinai Hospital, New York City.

Section of Anesthesiology

Parlor C, Hotel Statler

Dr. Sidney C. Wiggin, Waban, *Chairman*
Dr. Leo V. Hand, Newton, *Secretary*

Progress in Anesthesia in the Veterans Administration. Dr. Ralph M. Tovell, Hartford, Connecticut. Chief, Department of Anesthesiology, Hartford Hospital.

Section of Radiology

4th Floor, Hotel Statler

Dr. George Levene, Chestnut Hill, *Chairman*
Dr. Edward B. D. Neuhauser, Cambridge, *Secretary*

Encephalographic Studies of Patients with Head Injuries. Dr. Richard H. Schatzki, in collaboration with Dr. Donald H. Baxter and Dr. Charles E. Troland.

Section of Physiotherapy

Parlors D and E, Hotel Statler

Dr. Arthur L. Watkins, Boston, *Chairman*
Dr. Howard Moore, Boston, *Secretary*

A Sound Moving Picture on Rehabilitation Entitled "Accident Service." With a discussion by Dr. George C. Deaver, New York City, and Dr. Alexander P. Aitken, Boston.

THURSDAY AFTERNOON, MAY 23

Fifth General Session

Georgian Room

Dr. Frank R. Ober, *Chairman*
Dr. Harry Blotner, *Co-chairman*

2:00 *Some Current Trends in Obstetrics.* Dr. Samuel A. Cosgrove, Jersey City, New Jersey. Clinical professor of obstetrics, Columbia University Faculty of Medicine; medical director, Margaret Hague Maternity Hospital, Jersey City, New Jersey.

2:25 *Some Practical Aspects of Electroencephalography.* Dr. Randolph K. Byers, Boston. Associate visiting physician, Children's Hospital; instructor in pediatrics, Harvard Medical School.

2:50 *The Ulcer Problem.* Dr. Owen H. Wangensteen, Minneapolis. Chief, Department of Surgery, University of Minnesota Medical School.

3:15 *Skin Manifestations of Capillary Fragility: Their Diagnosis and Treatment.* Dr. Samuel M. Peck, New York City. Associate attending dermatologist, Mount Sinai Hospital, New York City. Dr. Alfred L. Copley, research associate, Department of Biology, New York University.

3:40 *Physical Rehabilitation of the Disabled.* Dr. George C. Deaver, New York City. Physician in charge of physical medicine, Bellevue Hospital; medical director, Institute for Crippled and Disabled.

4:05 *A Discussion of Some Debatable Questions in the Treatment of Heart Disease.* Dr. William D. Stroud, Philadelphia. Professor of cardiology, University of Pennsylvania Graduate School of Medicine; associate in medicine, University of Pennsylvania School of Medicine; cardiologist to the Pennsylvania Hospital.

4:30 *Blood Transfusions and Transfusion Reactions.* Dr. William Dameshek, Boston. Professor of clinical medicine, Tufts College Medical School; hematologist, Pratt Diagnostic Hospital.

MOTION-PICTURE PROGRAM

Salle Moderne

Wednesday, May 22

9:00– 9:40 *The Story of DDT.*
9:45–10:10 *Esophageal Diverticulum.*
10:15–11:00 *Hemothorax, Empyema and Specific Remedial Breathing Exercises* (courtesy of Dr. D. E. Harken).
3:00– 3:20 *Complete Neck Dissection.*
3:25– 4:05 *Combined Operation: Fusion of lumbosacral joint.*
4:15– 5:00 *Foreign Bodies in the Lung and Mediastinum* (courtesy of Dr. D. E. Harken).

Thursday, May 23

9:00–9:35 *Skin Grafting.*
9:40–10:10 *Aseptic Ileocolostomy.*
10:15–11:00 *Foreign Bodies in the Pericardium and Heart* (courtesy of Dr. D. E. Harken).
2:30– 3:00 *Cancer of the Female Breast.*
3:05– 3:25 *Subtotal Thyroidectomy.*
3:30– 4:00 *Physical Rehabilitation of the Disabled* (courtesy of Dr. George C. Deaver).

SCIENTIFIC EXHIBITS

Ballroom Assembly

BOOTH

S-13 *Council of Dental Health Program.* Massachusetts Dental Society.

S-14 *Modern Pharmacy.* Massachusetts Pharmaceutical Association.

S-15 *Blue Shield: The doctors' voluntary plan.* Massachusetts Medical Service.

S-16 *Enuresis: The use of cystourethrography in diagnosis.* Dr. M. Leopold Brodny and Dr. Samuel A. Robins.

S-17 *Anesthesia.* THE MASSACHUSETTS MEMBERS OF THE NEW ENGLAND SOCIETY OF ANESTHESIOLOGY. Exhibitor: Boston City Hospital.

S-18 *Anesthesia.* THE MASSACHUSETTS MEMBERS OF THE NEW ENGLAND SOCIETY OF ANESTHESIOLOGY. Exhibitor: Beth Israel Hospital.

S-19 *Anesthesia.* THE MASSACHUSETTS MEMBERS OF THE NEW ENGLAND SOCIETY OF ANESTHESIOLOGY. Exhibitor: Massachusetts Memorial Hospitals.

S-20 *Industrial Dermatology.* DEPARTMENT OF DERMATOLOGY, TUFTS COLLEGE MEDICAL SCHOOL.

S-21 *Massachusetts Study of Child Health Services.* AMERICAN ACADEMY OF PEDIATRICS. Exhibitor: Dr. Lendon Snedeker, deputy state chairman.

S-22 *Anesthesia and Thyroid Surgery.* THE LAHEY CLINIC. Exhibitors: Dr. U. H. Eversole and associates.

S-23 *Thiouracil in Thyroid Surgery.* THE LAHEY CLINIC. Exhibitor: Dr. Elmer C. Bartels.

S-24 *Military Plastic Surgery.* VALLEY FORGE GENERAL HOSPITAL. Exhibitor: Lieutenant Colonel Bradford Cannon, M.C., A.U.S., and Captain Joseph E. Murray, M.C., A.U.S.

S-25 *Tuberculosis Control.* HEALTH DEPARTMENT, CITY OF BOSTON (Frederick J. Bailey, M.D., Commissioner).

S-26 *Sterility.* FREE HOSPITAL FOR WOMEN. Exhibitor: Dr. John Rock.

S-27 *The First Two Weeks of Human Development.* FREE HOSPITAL FOR WOMEN, AND CARNEGIE INSTITUTION OF WASHINGTON. Exhibitor: Dr. Arthur T. Hertig.

S-28 *Blood Fractionation Program.* THE MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH.

TECHNICAL EXHIBITS

The booths without letters are in the Ballroom; those preceded by "S" are in the Ballroom Assembly; and those preceded by "M" are in the Mezzanine.

BOOTH

Abbott Laboratories 56
Alkalol Company 22
American Hospital Supply Corp. S-6
Ames Company, Inc. 44
Ayerst, McKenna & Harrison 19
Chester Baker, Inc. S-33
Baker Laboratories S-5
Best Foods, Inc. M-11
Bilhuber-Knoll Corp. 34
Ernst Bischoff Company M-6
The Borden Company 28
Brewer and Company, Inc. ..... 4 & 5
Bristol Laboratories, Inc. M-29
Buffington's, Inc. 39
Burroughs Wellcome & Company 23
Cambridge Instrument Company M-30
Camel Cigarettes ..... 11 & 12
Carnation Company M-12
Certified Milk Producers Association S-4
Children Incorporated 9
Ciba Pharmaceutical Products, Inc. S-7
Coca-Cola Company ..... 13 & 14
Crosbie-Macdonald 46
Davies, Rose & Company 40
Denver Chemical Mfg. Company M-24
Doho Chemical Company S-2
Eaton Laboratories, Inc. ..... M-25 & 26
J. H. Emerson Company S-12
C. B. Fleet & Company 45
Gerber Products Company 43
J. E. Hanger, Inc. 35
Hanovia Chemical Mfg. Company 51
H. J. Heinz Company 27
Hoffmann-La Roche, Inc. 29
The Hygeia Nursing Bottle Company M-21
Hynson, Westcott & Dunning, Inc. 17
The "Junket" Folks M-20
Kalak Water Co. of New York, Inc. S-9A
The Kelley-Koett Manufacturing Company 7
Kellogg Company M-5
Kidde Manufacturing Co., Inc. M-23
Lederle Laboratories, Inc. 57
Lee De Forest Laboratories 32
Libby, McNeil & Libby 20
Eli Lilly & Company 38
J. B. Lippincott Co. S-10
M & R Dietetic Laboratories 16
E. F. Mahady & Company ..... Stage
Mead Johnson & Company ..... 52 & 53
Medical Protective Company 21
Mellin's Food Company 31
The Mennen Company S-9
William S. Merrell Company S-11
The National Drug Company 1
Nestle's Milk Products, Inc. M-4
Nestle's Milk Products, Inc. Food Sales Dept. M-28
T. J. Noonan Company M-18
North American Philips Company 10
Parke, Davis & Company M-10
E. L. Patch Company 15
Pet Milk Sales Corporation 25
Philip Morris Company, Ltd., Inc. 49
Rare Chemicals, Incorporated M-22
L. & B. Reiner 33
Riedel-de Haen, Inc. 54
Sandoz Chemical Works, Inc. 42
Schenley Laboratories, Inc. S-8
Schering Corporation 41
G. D. Searle & Company 24
Sharp & Dohme, Inc. 50
Smith, Kline and French Laboratories 8
Spencer, Inc. S-3
E. R. Squibb & Sons M-7
Frederick Stearns & Company ..... 47 & 48
Surgeons & Physicians Supply Company 30
Tailby-Nason Company 18
Vaponefrin Company 6
Walker Vitamin Products, Inc. M-27
Westinghouse X-Ray Company ..... 2 & 3
White Laboratories, Inc. ..... 36 & 37
Winthrop Chemical Company, Inc. 55
Wyeth, Incorporated M-1, 2, 3
F. E. Young & Company 26
The Zemmer Company, Inc. M-19

CORRESPONDENCE

MILITARY MEDICINE

To the Editor: The writer of the editorial "Merger and Medical Service," which appeared in the March 28 issue of the *Journal*, has expressed some views which deserve further comment. It seems to me that, in advocating a merger of the medical services of all the armed forces, he has gone unnecessarily far out of his way to disparage military medicine as practiced during the recent international disagreement.

One's opinions on almost any subject are derived either from what he himself has experienced or from what he has read or heard of the experiences of others. If the gentleman who wrote this editorial served personally with the Medical Corps of the Army or Navy he has, of course, every right to express his views, however dim they may be, but if this is actually the case, I suggest that his experience differed considerably from that of the majority of medical officers. If his expressed opinions are based on hearsay, as he seems to imply, I submit that possibly he has listened too attentively to a vocal minority, many of whom have purely personal reasons for their dissatisfaction with military medicine.

Although I am not qualified to speak of medicine in the Navy or of Army medicine in the Asiatic-Pacific or Mediterranean theaters, forty-four months' experience as an Army medical officer in this country, England, France and Germany has brought me to the following conclusions, with which I believe that the vast majority of my former colleagues would agree:

Army medicine is in so many respects superior to civilian medicine that there is no legitimate basis for comparison. This is a sweeping generalization, but there are so many specific examples by which it can be substantiated that I hardly know where to begin. I should ask your editorialist to consider the incidence of typhus in the American army in the ETO; I should ask him what he thinks of the military policy of universal compulsory chest x-ray films on all personnel; I should ask him to cite a single civilian community where expert, free medical care, hospitalization and laboratory facilities are constantly available to anyone at any time, regardless of his social or economic status. Finally, I should ask this: If civilian medicine is so superior, why all the current agitation, from almost every quarter, to remodel it completely?

One frequently hears the statement, "Army medicine was fine, but only because so many civilian doctors joined the Army." The absurdity of this viewpoint becomes apparent when one considers that in time of war no department of the Army would be of much value if it were not for civilian soldiers. No one will maintain that Army medicine was perfect; we all saw situations that could have been improved (and usually were, in time), conditions requiring correction (they usually got it) and procedures that were carried out inefficiently (especially by inexperienced personnel, who later learned Army technics). In my own personal experience, I never saw a "snafu" of any magnitude that was not the direct or indirect result of some former civilian's attempt to do things in his own — and often highly original — fashion, rather than in the time-tested and experience-proved Army way.

I do not understand exactly what your writer means when he speaks of medical officers being "treated as enlisted men." The statement is too vague for comment. When he states that they were "ordered to do menial duties," I should again like more details. I, myself, never saw this happen. I know many a former major or colonel who is not now above helping his wife with the after-supper dishes; none ever washed a plate in the Army. I wonder what the "menial duties" were, and who gave the orders.

If the writer of your editorial would accompany me to any local civilian hospital he cares to designate, I shall undertake to point out to him defects in cleanliness, sanitation and administration that would not be tolerated in an Army installation. He will probably see gum-chewing ward attendants, dirty food handlers, smoking in kitchens, orderlies in need of shaves or haircuts, dusty floors, unnecessary noise, grime under the radiators — but why go on?

Finally, one should consider the care of the patient. In the last analysis, this will always depend for its quality on the character and qualifications of the doctors and nurses in attendance, and, to a lesser extent, on the amount and nature of the material, human as well as inanimate, that is available to assist them. Where but in the Army (or Navy) are to be found hospitals that combine all the following features: every nurse a graduate; every ward officer having completed at least one internship, and many, residencies; an almost unlimited supply of drugs, instruments, hospital furnishings and professional equipment of every type; a competent visiting staff actually in residence; expert consultants in every branch of medicine and surgery, always available; well trained, well disciplined ward personnel; laboratory, x-ray and other diagnostic facilities constantly at hand, with no restrictions on their legitimate use; the whole, at no cost to the patient, and in fact with economic considerations of any kind never a limiting factor.

Many of us who served with the forces deplore the currently fashionable tendency to indulge in the indoor sport of "knocking the Army." We resent it for a variety of reasons, but chiefly perhaps for these three:

We were there and know from our own observations that the over-all picture was superlatively good.

We do not understand how any civilian, however talented or however "vocal," can possibly be in a position to judge the quality of military medical care.

We observe, often to our own embarrassment, that the most virulent criticism almost invariably emanates from those who have purely personal grievances, whether real or imaginary, against the military system; one, perhaps, failed to get promoted, another was relieved of his command, a third is upset because "someone else got the Legion of Merit." This translation of personal and usually deserved misfortune into a blanket condemnation of the Army as a whole is as subversive and as unconstructive as it is in bad taste.

WINTHROP WETHERBEE, JR.
24 School Street
Boston

* * *

The editorial in question does not unqualifiedly condemn military medicine. It does, however, imply that mistakes were made and that much might be accomplished toward future improvement if these faults were acknowledged and attempts were made toward their correction. Unfortunately, the experiences of many of the medical officers, particularly those assigned to small medical units, were not so satisfying as those of Dr. Wetherbee and his associates.

It is extremely difficult and hazardous to compare military and civilian medical care, since the recipients of the former are controlled by a rigid system of discipline. On the other hand, it must be acknowledged that the majority of advances, particularly in peacetime, originate in the large teaching units, and the editorial suggests that one of the aims of a merged medical department might well be a closer affiliation with such units than has existed in the past. — ED.

REPORT OF MEETING

MASSACHUSETTS CENTRAL HEALTH COUNCIL

The annual meeting of the Massachusetts Central Health Council was held on April 9, 1946. At the business meeting, after reports for the year by the president and the secretary-treasurer, the following officers were elected for the ensuing year: president: Dr. George C. Shattuck; vice-president: Dr. Channing Frothingham; and secretary-treasurer: Mr. Arthur J. Strawson.

The business meeting was followed by a discussion entitled "Symposium on Group Practice."* The principal speaker was Dr. Kingsley Roberts, director of Medical Administration Service, Incorporated, New York City.

Dr. Roberts approached the subject from the angle of better distribution of good medical care. He believed that, however financed, medical care should be organized on a prepayment basis; that the unit for care should be the family and not the individual; that the plan should include care in the home as well as in the hospital; and that the use of preven-

*The opinions expressed are those of the speakers. The Massachusetts Central Health Council has taken no position for or against group practice.

tive methods should be an essential part of the plan. To these ends, physicians should know more about socioeconomic matters and about how to persuade patients to take advantage of the periodic physical examination and of other means of conserving health. He added that some units offering group practice are well organized and that others are not. Certain of them offer only diagnostic, consultative or specialized services. Others offer service of a general character. For broad medical coverage, co-ordination of the work of the staff by a person who understands preventive as well as curative medicine is essential. Research should also be a function provided for in the budget.

The development of group practice, in the opinion of Dr. Roberts, is necessary to keep down cost, to promote the efficient distribution of medical care and to encourage the systematic application of preventive methods.

For the future, Dr. Roberts envisioned health centers in small communities, to be served by groups of physicians. A number of such health centers should be affiliated with a teaching and hospital unit in the nearest large city, and a flow of personnel back and forth between the teaching unit and the health centers should be provided for. Such a plan would not only improve medical service in small communities but it would also provide the participating physicians in such communities with an adequate income and educational advantages.

Dr. Roberts then contrasted several types of group practice that are now in operation and said that many medical veterans have been impressed with the advantages of group practice as it exists in the Army and have been asking advice about how to form or to join a group for practice in civil life.

The formal discussion was opened by Dr. Elmer S. Bagnall, of Groveland. Dr. Bagnall deplored the fact that discussions of possible changes in medical organization too often are characterized by controversialism. He believed that discussions aimed at expanding the areas of agreement should continue to be held and that this approach would prove profitable. Although not a member of a medical group, Dr. Bagnall said that the problems of organizing group medicine are worthy of study. Because people in a democracy resent regimentation and insist on the right to make mistakes, he thought that change should be gradual and by evolution. As a means of further study of medical problems on a community basis, Dr. Bagnall added that the formation of local health councils should be further stimulated, and he pointed out that the Massachusetts Medical Society has been discussing a two-way system of arranging for hospital care through central hospitals and subsidiary hospitals. The importance of the general practitioner as the co-ordinating link between the patient and the various specialists was stressed.

Dr. Fallon, of Worcester, who has been practicing in a group for many years, emphasized the ease of consultation within the group and the fact that diverse opinions can be expressed with the utmost freedom. Usually, the patient gets the benefit of at least two viewpoints. Laboratory and x-ray examinations are facilitated. When the cost of x-ray examination exceeds a specified figure, the group bears the additional expense. The members of the group, although specially qualified in certain fields, perform general work as well, and the internist gets a more equitable remuneration than is usual when surgery is required.

He added that there are many obstacles to success in group practice. Consequently, many groups have survived for only a few years. Dr. Fallon said that he did not wish to see group medicine replace the family doctor, who alone can have intimate knowledge of the patient's background.

Dr. Parkhurst, of Beverly, a member of a medical group, expressed interest in the plans outlined by Dr. Roberts but thought that it would be a long time before such a plan could be put into successful operation. He and other members of his group see patients not only in the office but also in the home. The patient chooses his physician within the group, but the group does not attempt to replace the general practitioner. Since a group is only as good as the men behind it, it should be built on the basis of ability to give good service rather than be formed from the physicians who happen to reside in the same locality. In certain instances it may be necessary to bring in outsiders having special qualifications. He added that a well organized group can not only provide improved services to a community but it may also become the nucleus of an efficient hospital staff.

Dr. Lendon Snedeker, of Boston, who at present is engaged in a fact-finding survey of health services for children in Massachusetts sponsored by the American Society of Pediatrics, said that Dr. Roberts's tentative plan is not unlike a scheme that is now in operation in Sweden, with private practice as an overlay. "Why, indeed," he said, "cannot a general practitioner who is the family counselor be the key man in a group?"

In closing, Dr. Roberts pointed out that his general plan can be and should be adapted to local needs and that he too was convinced of the need for the general practitioner.

BOOK REVIEWS

Neuro-Ophthalmology. By Donald J. Lyle, M.D. 4°, cloth, 395 pp., with 234 illustrations. Springfield, Illinois: Charles C Thomas, 1945. $10.50.

This book, the first American text to cover overlapping territory in the fields of neurology and ophthalmology, should prove a useful reference summary for practitioners and teachers of neurology and ophthalmology. Material for the text has been derived from 130 case histories, in all but 4 of which the clinical diagnoses were verified anatomically. Seventy-one pages are given to over seventeen hundred references, alphabetically arranged. There are 234 black-and-white figures, with 529 separate illustrations, diagrams or photographs.

Ophthalmologists particularly should be grateful to the author for portraying vividly by pen and picture the related neurologic and ophthalmologic aspects of intracranial lesions and for correlating the same by collateral anatomical diagrams and photomicrographs, x-ray photographs, visual fields and fundus photographs. One of the outstanding virtues of this work is that it brings to the ophthalmologist a neurologic point of view and a better understanding of visuomotor and visuosensory pathology.

Although the reviewer has only admiration for this important work, and faith that revision for the second edition will iron out the few rough spots, he wonders how the publisher could fail to include much of the preface and the first ten pages of Chapter I, whether the author would not consider it helpful to clarify the signs in supranuclear, nuclear, infranuclear and internuclear lesions affecting the third, fourth and sixth cranial nerves, and whether half the photographs of fundi should be replaced by more informative diagrams or photomicrographs of significant intracranial lesions.

Clinical Roentgenology of the Digestive Tract. By Maurice Feldman, M.D. Second edition. 8°, cloth, 769 pp., with 551 illustrations. Baltimore: The Williams and Wilkins Company, 1945. $7.00.

This book is largely a review of the older literature, giving major points in roentgenologic diagnosis and stressing their clinical application. For the student of roentgenology or of gastroenterology too many varieties of lesions, with rather questionable proof, are stressed. For the practitioner of either roentgenology or gastroenterology, however, the book should be an excellent reference source, so long as common sense is used in interpreting the extremely uncommon or doubtful types of lesions.

An excellent index and bibliography are included. The latter is divided, being appended at the end of each section, which makes it of particular value because of its ready accessibility.

Peripheral Nerve Injuries: Principles of diagnosis. By Captain Webb Haymaker, M.C., A.U.S., and Major Barnes Woodhall, M.C., A.U.S. 8°, cloth, 227 pp., with 225 illustrations. Philadelphia: W. B. Saunders Company, 1945. $4.50.

This book covers a field already well cultivated. The particular value of this publication lies, however, in the expert condensation and the unusual illustrations. The text is accurate and dependable, but the student or physician will gain most from a study of the photographs and drawings that accompany it. No previous publication on this subject is more clearly illustrated. Each maneuver of testing motion is not only described, but all phases of it are shown in the pen sketches. The book should appeal to a wide audience,

particularly medical students and physicians who practice neurology and neurosurgery. Printed on excellent paper, with clear type and a good binding, this book deserves the highest recommendation. It was widely distributed by the United States Army.

The Falling Sickness: A history of epilepsy from the Greeks to the beginnings of modern neurology. By Owsei Temkin, M.D. 4°, cloth, 380 pp., with 7 illustrations. Baltimore: Johns Hopkins Press, 1945. $4.00.

Dr. Temkin, who is an associate professor in the Department of History of Medicine at Johns Hopkins University, writes on the history of epilepsy from the time of the earliest reference by the ancient Greeks to 1880, when the effect of the work of Hughlings Jackson and Charcot became incorporated into medical thought. Naturally, much of the material, particularly that dealing with the earlier periods, is extracted from works dealing with speculations found in the opinions of laymen philosophers and theologians, as well as those of physicians. The disease was closely associated with magic, lunacy, theology and the literature surrounding such vague subjects as the supernatural and the occult. It was not until the beginning of the nineteenth century that epileptic patients began to be hospitalized and carefully observed. Charcot separated out the epileptic manifestations due to hysteria, and Jackson, the symptoms resulting from a focal lesion in the brain.

Dr. Temkin has covered the whole story in a skillful manner. References are given to every statement of fact. His style is meticulous, and the chapters are supplemented by an extensive bibliography and an excellent index. The book is a credit to American medicine in the historical field and should be of use to all historians and those physicians who have to deal with convulsive disorders.

The Modern Medical World: Portraits and biographical sketches of distinguished men in medicine. By Solomon R. Kagan, M.D. 8°, cloth, 223 pp., illustrated. Boston: Medico-Historical Press, 1945. $6.00.

In this volume the author presents a series of admirable biographical sketches and portraits of three hundred and twenty medical men distinguished for their notable contributions to science from the beginning of the nineteenth century. A large proportion of the portraits are reproduced for the first time. The names are arranged not alphabetically but chronologically under groups of men working in identical fields. In this collection and publication Dr. Kagan makes a valuable contribution to the record of modern medical history.

Microbial Antagonisms and Antibiotic Substances. By Selman A. Waksman, M.S. and Ph.D. 8°, cloth, 350 pp., with frontispiece. New York: The Commonwealth Fund, 1945. $3.75.

The chasms separating many of the fields of abstract and applied science are often as wide as that between science in general and the layman's understanding thereof. This is a book bridging one such void between the field of soil and water microbiology, where microbial antagonism is an old story, and practical medical science, where antagonism was, until recently, a forgotten one.

The first two chapters discuss soil and water micro-organisms and their interrelations. Taken as a group, these forms of life make up the oldest of all living associations. It soon appears that they also have their parasitic parvenues. In the third chapter the field narrows to a consideration of these. The medical reader may be somewhat startled to find that the author apparently favors classifying one of medicine's venerable D.A.R.'s, the leprosy bacillus, as a "facultatively parasitic soil organism." The field still further narrows, however, to include only those parasites which have developed mass-murder methods of a high order against their fellow microbes. The fourth chapter, replete with research suggestions and possibilities, outlines methods of finding and isolating these antagonistic organisms and of identifying and assaying their products.

The next five chapters cover the known antagonists and the antibiotic agents of five broad groups of micro-organisms — bacteria, actinomycetes, higher fungi, protozoa and viruses. The tenth chapter lists the thirty-odd antibiotic agents of note in the five broad groups and outlines what is known of their chemical composition. The eleventh discusses antibiotic action and distinguishes it from antiseptic action. The remaining chapters take up present applications and future possibilities.

The applied scientist usually likes his data in handy-reference form and may regret that each antibiotic substance is treated in so many places in the book. Many such readers could benefit from an example of thorough, if apparently digressive, treatment. Good balance is maintained in the handling of the various substances — none, not even the author's favorite, receiving undue consideration at the expense of the others.

The book provides the interested beginner with the technics and basic background needed for a start. At the very least it should give the conventional bacteriologist with his mania for pure-culture study something more exciting to look for than the discard-bucket when he finds bacterial and mold contaminants on his plates. The sixty-page bibliography will keep him from rushing into print with his first discovery.

NOTICES

ANNOUNCEMENTS

Dr. George L. Maltby announces the opening of an office at 29 Deering Street, Portland, Maine, for the practice of neurological surgery.

Dr. Philip S. Marcus, having returned from military service, announces the reopening of his office for the practice of anesthesiology at 270 Commonwealth Avenue, Boston.

Dr. H. Herman Shuman announces his return from military service and resumption of the practice of pediatrics at 34 Maple Street, Springfield.

Dr. Garrett L. Sullivan, having returned from military service, is resuming the practice of ophthalmology at 101 Bay State Road, Boston.

NEW ENGLAND SOCIETY OF PHYSICAL MEDICINE

The regular meeting of the New England Society of Physical Medicine will be held at the Ring Sanatorium and Hospital, Arlington, on Thursday, May 16, at 8 p.m. Dr. John G. Trump will speak on the topic, "High-Voltage X-Rays and Cathode Rays for Cancer Therapy."

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Thursday, May 16

Friday, May 17

*9:00–10:00 a.m. Clinicopathological Conference. Drs. Stanley Bradley and H. E. MacMahon. Joseph H. Pratt Diagnostic Hospital.

*10:00 a.m.–12:00 m. Medical Staff Rounds. Peter Bent Brigham Hospital.

Monday, May 20

*12:15–1:15 p.m. Clinicopathological Conference. Peter Bent Brigham Hospital.

Tuesday, May 21

*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

Wednesday, May 22

*9:00–10:00 a.m. Hyperadrenocorticism. Dr. Robert H. Williams. Joseph H. Pratt Diagnostic Hospital.

*10:30–11:30 a.m. Medical Clinic. Isolation Building Amphitheater, Children's Hospital.

*12:00 m. Clinicopathological Conference. (Children's Hospital.) Amphitheater, Peter Bent Brigham Hospital.

*2:30–4:00 p.m. Combined Clinic by the Medical, Surgical and Orthopedic Services. Amphitheater, Children's Hospital.

*Open to the medical profession.

March 15–September 15. Boston University Course for Discharged Medical Officers. Page 240, issue of February 14.

April 1–June 1. Intensive Course in Ophthalmology. Page 240, issue of February 14.

May 6–11. American Board of Obstetrics and Gynecology. Page 458, issue of March 28.

(*Notices continued on page xvii*)